Chapter 10

The Women with Multiple Trigger Factors

Vincent T. Martin, MD

Case History

A 35-year-old Caucasian female presents to our office with a chief complaint of "headache." She has seen mul-tiple physicians (eg, primary-care physicians, neurologists, and one headache physician) in the past, with minimal improvement in her headaches. She states that no one has adequately explained what is causing her headaches, and she will not quit until she finds the answer.

Her more severe headaches are described as "throbbing." The pain originates in the neck and then radiates to the unilateral frontal, temporal, parietal, and occipital regions. The average pain intensity of these headaches (on a scale of 0 to 10) is 8, and they last for 24 to 48 hours. These headaches may cause her to miss work and social events. They are associated with nausea, but no vomiting, photophobia, or phonophobia. She denies neurologic symptoms such as scintillating scotoma, hemiparesis, hemisensory loss, ataxia, diplopia, or vertigo. Her headaches first began at menarche and initially occurred 1 to 2 days each month, primarily around each menstrual period. At age 16 years, she suffered a "whiplash" injury secondary to a motor vehicle accident, which seemed to increase the frequency of her severe headaches to 4 to 6 days per month. They remained at this frequency until she underwent a divorce 4 months ago, at which time the frequency of her headaches increased to 10 to 14 days per month. She reports being under tremendous "stress."

Her less severe headaches occur in the bilateral occipital region and occur 18 to 20 days per month. They have an average pain intensity of 3 and cause no significant disability to the patient. They are not associated with nausea, vomiting, photophobia, or phonophobia. She denies neurologic symptoms with these headaches. The headaches first arose during her teenage years and have not changed in frequency, severity, or duration.

The patient thinks that she has multiple trigger factors for her headaches. Her most significant trigger is her menstrual periods. Her more severe headaches predictably occur 2 days before the onset of menstrual bleeding, and last for 3 days during the placebo week of her oral contraceptive. Other triggers include red wine, flickering lights, weather changes, aspartame, and chocolate. Her headaches also seem to worsen in the spring and fall, which she attributes to a worsening of her allergies. She states that with the recent divorce, she has had depressed mood, lack of interest in pleasurable activities, and lack of appetite. She has difficulty falling asleep and maintaining sleep. She gets approximately 5 to 6 hours of sleep per night.

Her past medical history is significant for allergic rhinitis and asthma since childhood. She has had two past childbirths (at 22 and 26 years of age), and her headaches significantly improved during the second and third trimesters, but her headaches significantly worsened for 1 to 2 weeks after each delivery. Her medications included a beta-2 agonist inhaler, an inhaled steroid, a triptan, and a monophasic birth control pill containing 20 mg of ethinyl estradiol. She did not take over-the-counter medications to abort her migraine headaches. Her family history includes a mother and sister with migraine headache. The patient smokes two packs of cigarettes per day and drinks 3 to 4 alcoholic beverages per week. She also consumes four 8-oz cups of brewed coffee per day. She denies drug use. She works 60 hours per week as a lawyer. Her physical examination revealed a height of 5 feet 7 inches and a weight of 200 pounds, but was otherwise unremarkable. A review of systems was positive for loud snoring, daytime somnolence, and morning headaches.

The patient had a negative magnetic resonance imaging result of the brain 3 months ago. She has tried a number of preventative medications including beta-blockers, cal-

cium channel blockers, tricyclic antidepressants, divalproex sodium, gabapentin, and topiramate. None of these medications has been particularly effective, and many caused intolerable side effects. She is not interested in trying new migraine preventive medications, since they are "just masking the true cause of the headaches." She wants the treating physician to "please help [me] find the 'cause' of [my] headaches."

Questions on the Case

Please read the questions, try to answer them, and reflect on your answers before reading the author's discussion.

- What is the most appropriate diagnosis or diagnoses for this patient's headache disorder?
- What is the "cause" of this patient's headache disorder?
- What are the potential headache triggers in this patient?
- How does one identify a migraine or headache trigger in this patient?
- How can we manage the headache triggers in this patient?
- Describe the prevalence and clinical characteristics of migraine triggers (eg, the time required after exposure to produce a headache, or the likelihood of a headache after exposure to a known trigger).

Case Discussion

Diagnosis

The patient's headaches meet the diagnostic criteria for both episodic migraine without aura and chronic tension-type headache (2004 International Headache Society [IHS] criteria 1.1 and 2.3, respectively). These two diagnoses are discussed below.

The most common mistake made when interviewing a patient is to ask the following question: "Describe your headaches." The problem with this question is that > 50% of patients will present with more than one headache disorder simultaneously. Therefore, asking such a question will elicit mixed symptoms from all of the headache disorders, making the individual diagnoses difficult. More appropriately, one could ask the patient, "Do you have more than one type of headache? If yes, describe your more severe or disabling headaches." This interviewing technique was done in this patient and allowed us to tease out both diagnoses.

This patient's more severe headaches lasted 24 to 48 hours and were throbbing, severe, unilateral, and associated with nausea. They met the diagnostic criteria for episodic migraine without aura. The fact that her headaches first arose in the neck might dissuade some from a diagnosis of migraine headache in favor of a diagnosis of "cervicogenic headache." However, neck pain is quite common in migraine headache, occurring in 75% of all patients. Her less severe headaches were bilateral, mild, lacked associated symptoms, and occurred 18 to 20 days per month. Therefore, these headaches met the diagnostic criteria for chronic tension-type headaches.

The patient suffers from obesity and has symptoms of sleep apnea, such as morning headaches, daytime somnolence, and loud snoring. One should consider sleep apnea, as well as pseudotumor cerebri, in the differential diagnosis of her headache disorders. She may deserve a lumbar puncture to exclude the latter diagnosis, even in the absence of papilledema.

Establishing a correct diagnosis in this patient is important since some triggers may only provoke migraine headache, while others may provoke headaches of all kinds. Also, the frequency of headache may play a role in this case. Our patient suffers from chronic daily headache (> 15 days per month with headache). Patients with more frequent headaches often report a greater number of trigger factors. It is unknown whether patients with chronic daily headache are more susceptible to triggers or simply that they are more likely to report them.

Headache Triggers

Patients frequently confuse what "causes" migraine with what "triggers" migraine. The patient was told that she had inherited a nervous system that was "hypersensitive" to the triggers in the environment. This "hypersensitive" nervous system was the cause of her migraine headaches, but exposure to triggers may have led to a worsening of her headaches.

Four triggers, in particular, may have played an important role in modulation of her migraine headaches throughout her life. First, the patient experienced a "whiplash" injury to her neck at a young age, which may have led to the initial worsening of her headaches as well as the neck pain that she experiences with her headaches today. Second, she may have a hormonal trigger for headaches. Her headaches worsened around her menstrual period, improved during the second and third trimesters of pregnancy, and worsened immediately after each pregnancy. This suggests that her migraines are "hormonally sensitive." Third, she reports a lot of stress with a recent divorce and a subsequent worsening of her headaches. Stress is one of the most common trigger factors reported by patients. The patient may also be experiencing depression as a result of this stressful life event. Fourth, her caffeine consumption may have led to caffeine withdrawal headaches, thus increasing the frequency of her headaches.

She also has a history of atopy and reports that her headaches worsen during the spring and fall, secondary to allergies. Although allergic rhinitis is not a universally

accepted trigger factor, some studies have found an association between allergic rhinitis and migraine headache. Other potential migraine triggers in this patient include wine, chocolate, aspartame, flickering lights, and smoking.

Identification of triggers may come from self-report by the patient (as occurred in this patient), or from use of a headache diary. Use of a headache diary may identify new triggers as well as confirm past triggers. Patients are asked to record the severity of their headaches (0 to 10 scale) in the morning, afternoon, and evening. All foods and beverages that are consumed during breakfast, lunch, dinner, and snacks are also listed in the diary. A trigger may exist if a new headache begins or an existing headache worsens within 24 hours after exposure. A trigger may be classified as a "definite trigger" if in greater than 50% of the time a headache begins or an existing headache worsens after exposure to the trigger, a "possible trigger" if there is a 25 to 50% association, and an "unlikely trigger" if there is < 25% association. Therefore, it would be reasonable to give this patient a headache diary. To interpret a headache diary, one must understand the clinical characteristics of triggers (eg, the time to provoke a headache after exposure, or the likelihood to provoke a headache after exposure), which will be discussed below.

Management Strategies

This patient has tried a number of migraine preventative medications without success, but she has never attempted a treatment regimen that attempts to modulate factors that may actually trigger her headaches. The main management questions to be answered in this case are as follows:

- Is there a need for trigger factor modulation in this patient?
- How does one prioritize which trigger factors to modulate?
- After prioritizing, how does one modify these individual triggers?
- Should the patient be placed on a particular diet to prevent migraines?
- How long does it take to see an improvement in the headaches after modulation of the triggers?
- Are there any lifestyle changes that should be recommended to the patient to prevent headaches?

Clearly there is a need for trigger factor intervention in this patient. She refuses preventative medications and has a desire to address the "cause" of her headaches. Also, her migraine headaches are frequent (occurring 10 to 14 days per month) and disabling (causing her to miss work and social events).

This patient has a myriad of headache triggers, and it may not be practical to withdraw all of them. Therefore, some prioritizing of triggers is necessary. Behavioral and biologic triggers (eg, menstruation, stress, emotions, fasting, and sleep disturbances) are more commonly self-reported by patients as potential triggers than the exogenous triggers (eg, food, beverages). In the author's experience, the behavioral and biologic triggers have had a greater propensity to produce long-lasting changes in the headaches pattern, while the exogenous triggers have more of a short-lasting effect. This may be secondary to the fact that the patient is more consistently exposed to behavioral and biologic triggers, or alternatively, that these endogenous triggers are more potent. The one exception to the rule is the exogenous trigger, caffeine, which is one of the most potent and underrecognized triggers for headache. Based on this hierarchy, we will try to modify the following triggers in this patient: stress, emotional, hormonal, sleep disturbances, and caffeine. Management of the individual triggers will be discussed below.

Stress appears to be the one trigger factor that is most closely related to the recent worsening of her headaches. She has undergone a recent divorce, works 60 hours per week, and is responsible for the care of two children! Interestingly, stress is the most commonly self-reported headache trigger by migraine patients. The minor stresses of daily life are more predictive of headache than the major life events (divorce, death). The stressor generally occurs on the same day as the headache, or precedes it by 1 to 3 days. Reducing stress in this patient will not be easy, as many of her stressors cannot be changed. However, we may be able to modify how she responds to the stressors by recommending that she see a psychologist for biofeedback and relaxation techniques.

The patient may also have an emotional trigger for her headaches. The patient appears to manifest symptoms of a major depression (depressed mood, decreased interest in pleasurable activities, lack of appetite, lack of sleep). Depression is very prevalent, occurring in 25 to 80% of individuals with chronic daily headache. Although selective serotonin reuptake inhibitors (SSRI's) have not been convincingly shown to be effective in patients with episodic migraine, they have been effective in patients with chronic daily headache. Therefore, it might be reasonable to give this patient a trial of an SSRI or other antidepressant.

A hormonal trigger is likely in this patient, as she reports the onset of her migraine headaches 2 days before the onset of menstrual bleeding during the placebo week of her oral contraceptives. Therefore, she suffers from what is classified in the 2004 IHS classification appendix as menstrually related migraine, which are migraines that predictably occur 2 days before to 3 days after menstruation as well as during other times of the month. Menstrually related migraines likely result from the withdrawal of estrogen that occurs at the time of menstruation, either during a natural menstrual period or during the placebo

week of oral contraceptives. The frequency of her menstrually related migraines can be reduced by eliminating the falls in estrogen that occur during the placebo week of her oral contraceptive. One strategy would be to eliminate the placebo week from the first three packs of monophasic oral contraceptives (triphasic pills are not recommended), and administer it only after the fourth pack. She would only menstruate once every 3 months, and thus menstrual migraine would be reduced by 75%. Another strategy would be to administer a separate estrogen preparation (conjugated estrogens or transdermal estradiol-β) during the placebo week of the oral contraceptive. The patient would still menstruate, but the perimenstrual falls in estrogen would be reduced, as well as the menstrual migraine. It should be mentioned that the long-term safety of either of these approaches has not been established.

The patient has a sleep disturbance, which could be exacerbating her migraines. It is uncertain if the sleep disturbance is a primary disorder or is secondary to disorders such as depression or sleep apnea. Since she is being started on an SSRI, this will allow us to see if her insomnia improves with therapy for depression. With her symptoms of sleep apnea, it would be reasonable for her to undergo polysomnography. If her sleep does not improve with the above measures, then it would be reasonable to give her a sedative hypnotic. She should also practice good sleep hygiene, avoiding caffeinated beverages or exercise within 4 hours of bedtime and daytime naps.

Daily consumption of caffeine (in the form of a beverage or contained in a pill) likely represents one of the most important reasons for conversion from an episodic to a daily headache pattern. Caffeine withdrawal headaches begin within 24 to 48 hours after cessation of caffeine consumption and generally last for 1 to 6 days. In fact, withdrawal symptoms can develop after abrupt cessation in those who consume as little as 100 mg per day. Our patient consumed approximately 480 mg per day (four cups with 120 mg of caffeine per 8-oz cup of brewed coffee). Therefore, it would be recommended to gradually decaffeinate herself over a 4-week time period. In the author's experience, a more rapid taper may lead to worsening headaches.

The patient also reported exogenous triggers such as allergic rhinitis/allergies, flashing lights, smoking, and dietary triggers (discussed below). In the author's experience, migraine headaches may be exacerbated by allergic rhinitis, although this is quite controversial. Since treatment of allergic rhinitis with nasal steroids and antihistamines is fraught with few side effects, it would be reasonable to treat the allergic rhinitis during peak allergic seasons (spring and fall) to see if it helps the headaches. Bright and flashing lights are a recognized trigger for some patients, and therefore, their avoidance would be recommended. Using sunglasses during bright sunlight would be advised. Smoking is a controversial trigger, but studies suggest that migraineurs who smoke may have more severe headaches. Smoking cessation would be a good long-term goal for this patient, but would not be recommended initially with all of the other modifications and treatments mentioned above. Recommending too many interventions at the same time could overwhelm the patient.

Dietary triggers are reported by less than 30% of migraine patients and encompass numerous foods and beverages (Table 10-1). This represents a great number of foods and beverages, making it difficult and likely unhealthy to eliminate all of these triggers simultaneously. Also, studies of migraine diets have not demonstrated efficacy in the prevention of migraine headaches. Therefore, a migraine diet was not recommended for this patient. Ideally, dietary triggers should be identified through a headache diary. In our case, the patient had previously identified dietary triggers such as aspartame, chocolate, and red wine, and as a result, these should be avoided.

The time-course to improvement after withdrawal of an individual trigger factor, for this patient, likely depends on the trigger and the frequency of exposure. Triggers with intermittent or infrequent exposure will likely only provoke a headache shortly after exposure for 1 to 3 days. Therefore, their avoidance will completely eliminate the headaches. For example, if one prevents the perimenstrual falls in estrogen in this patient, then menstrual migraine will not occur or will become drastically attenuated. However, triggers with daily or frequent exposure, such as caffeine consumption, stress, sleep disturbances, or depression, may take longer to improve. For example, it may take 1 to 3 months to see an improvement after cessation of caffeine consumption or treatment of depression.

Healthy lifestyle practices should be recommended to this patient as well. Triggers such as stress, sleep disturbances, fasting, and bright lights are sufficiently common in the migraine population to recommend the following healthy lifestyle practices:

- Maintain regular sleep hygiene.
- Eat regularly and avoid fasting.
- Minimize stress through lifestyle changes.
- Exercise and engage in relaxation techniques.
- Avoid bright or flashing lights.

Case Summary

The patient was a 35-year-old woman with a 20-year history of migraine with aura and chronic tension-type headache, who reported an increased frequency of migraine headache in the prior 4 months. Her physical examination was normal, and she had negative neuroimaging. She has tried a number of past preventatives without success and

Table 10-1. Dietary Triggers for Migraine Headache

Foods	Examples
Alcohol	Wine, beer
Tyramine-containing foods	Cheese, wine, beer, smoked or pickled fish, nonfresh meats, broad beans, sauerkraut, dry sausage, yeast extract
Nitrate-containing foods	Cured meats, bacon, sausage, frankfurters, hot dogs, ham, luncheon meats
Monosodium glutamate-containing foods	Frozen foods, canned or dry soups, salad dressings, processed meats, tomato or barbecue sauce, snack foods
Fruits	Oranges, grapefruit, bananas
Aspartame-containing foods	Some beverages, desserts, snacks, sweeteners

has no interest in further preventatives since they treat the symptom and not the "cause" of her headaches. The patient reported a number of potential triggers for migraine headache including stress, depression, menstruation, sleep disturbances, caffeine consumption, allergic rhinitis, smoking, and diet. Presented with such a vast array of triggers, we must first prioritize the triggers, as it would be impractical to modify all the triggers simultaneously. Management of her headaches must focus on avoidance or modulation (possibly with medications) of these trigger factors. This represents a different kind of therapeutic approach than traditionally used in medicine. In the past, physicians have primarily treated the consequences of the disease, not the triggering event (eg, abortive or preventative therapy for migraine really does not address what led to the headache). Yet the question remains: "Does modulation of trigger factors make a difference?"

Overview of Migraine Trigger Factors

Definition

A migraine trigger is any factor that, upon exposure or withdrawal, leads to development of a migraine headache. Migraine trigger factors may be categorized as behavioral, environmental, infectious, dietary, chemical, or biologic (Table 10-2).

Pathophysiology

The pathophysiology of migraine triggers remains largely speculative. Triggers could act peripherally at the level of the dural blood vessel or trigeminal nerve, or centrally at the level of the trigeminal nucleus caudalis, thalamus, limbic system, brainstem modulatory pathways (locus coeruleus, dorsal raphe), or cortex. Interestingly, many of the triggers could potentially modulate one or more of the following three neurologic pathways:

1. Serotonergic (wine, weather changes, caffeine, menstruation, stress, fasting, smoking, sleep disturbances)
2. Noradrenergic (tyramine, phenylethylamine, caffeine, menstruation, chocolate, stress, sleep disturbances)
3. Nitric oxide (nitrates, monosodium glutamate, histamine, wine, smoking)

Prevalence

Patient surveys in subspecialty clinics and the general population have ascertained the prevalence of migraine trigger factors (Table 10-3). The prevalence of self-reported trigger factors is higher in those studies recruiting patients from subspecialty clinics than in those from the general population. This could be secondary to several factors:

- Subspecialty patients may be more susceptible to migraine triggers.
- Subspecialty patients may be more educated on migraine triggers and more likely to report them.
- Subspecialty patients could search harder to remember migraine triggers (recall bias).

Table 10-2. Categories of Migraine Triggers

Behavioral
Fasting
Emotions
Sleep disturbances
Stress
Exercise
Dietary
Caffeinated beverages
Alcoholic beverages
Aged cheeses
Chocolate
Ice cream
Environmental
Bright light/visual stimuli
Odors
Weather changes
Cigarette smoke
Chemical
Monosodium glutamate
Tyramine
Nitrates
Aspartame
Infectious
Upper respiratory infections
Biologic
Hormonal/menstruation

Adapted from Pryse-Phillips WE, Dodick DW, Edmeads J, et al. Guidelines for the nonpharmacologic management of migraine in clinical practice. CMAJ 1997;159:47–54.

Table 10-3. Prevalence of Migraine Trigger Factors

Trigger Factor	Ulrich*	Van Den Bergh*	Chabriat*	Turner*	Scharff†	Robbins†	Peatfield†
Stress/post-stress	36%	49%	42%	51%	72%	62%	—
Menstruation	—	8%	32%	54%	68%	50%	—
Changes in sleep	—	—	—	38%	52%	31%	—
Fasting	—	—	—	56%	45%	40%	—
Weather	—	7%	35%	52%	46%	43%	—
Food	—	44%	36%	22%	—	30%	—
Smoking	9%	4%	2%	—	—	26%	—
Chocolate	2%	23%	—	—	22%	—	19%
Alcohol	—	—	—	—	35%	—	29%
Caffeine	—	—	—	—	14%	—	—
Cheese	—	—	—	—	9%	—	18%
Monosodium glutamate	—	—	—	—	13%	—	—

Adapted from Martin VT. Toward a rational understanding of migraine trigger factors. Med Clin North Am 2001;85:911–41.

Includes women and men as well as migraine with and without aura; represents the self-reported prevalence or migraine trigger factors.

*Population-based study.

†Specialty-clinic-based study.

Clinical Characteristics

Identification of migraine triggers requires knowledge of their clinical characteristics. The following five questions relate to the clinical characteristics of migraine triggers:

- How long does it take for a trigger factor to provoke a headache?
- Are all trigger factors equally likely to provoke a migraine headache?
- Does susceptibility to one trigger make a patient more vulnerable to other triggers?
- When exposed to two triggers simultaneously, is one trigger more likely than another to result in a headache?
- How frequently does exposure to a known trigger lead to migraine headache?

The latency period of a trigger to provoke a migraine headache varies depending on the trigger, but migraine generally develops within 24 hours after exposure. Randomized controlled trials have demonstrated a latency period of 3, 12, and 22 hours for red wine, phenylethylamine, and chocolate, respectively.

Trigger factors may vary in their ability to provoke migraine headache. In other words, "all trigger factors may not be created equal." Generally, the behavioral and biologic triggers are more commonly self-reported by patients than the dietary and chemical triggers. This could be secondary to the fact that behavioral and biologic triggers are more clinically evident than exogenous triggers, or simply that the endogenous triggers are more potent. The author favors the later explanation. Of the dietary triggers, alcohol is the most frequently reported.

There seems to be a clustering of headache triggers in some patients. For example, Van den Bergh and colleagues found that patients with a major trigger such as stress, alcohol, or menstruation were more likely to report other triggers. Turner and colleagues reported that those patients with menstruation as a trigger were more likely to report fasting or fatigue as a trigger. Overlap of self-reported triggers does not prove that susceptibility to a single migraine trigger makes a patient more vulnerable to another trigger. Individuals reporting triggers may have been educated on them and thus more likely to report multiple triggers.

Synergism may exist between triggers. Nicolodi and Sicuteri found that wine was a trigger for migraine headache only during stressful times. Holm and colleagues found that stress was a trigger for migraine headache only during the week before menstruation. Therefore, some triggers may only provoke migraine when they coexist with another trigger. In other cases, both triggers could provoke migraine, but the likelihood of migraine may increase if two triggers occur together.

Triggers may not produce a migraine headache with each exposure. Randomized controlled studies suggest that migraine headache may occur after 42% of exposures for chocolate, and 82% of exposures for red wine in those susceptible. This characteristic, the incomplete penetrance of the clinical expression of the trigger-migraine coupling, in particular, must be appreciated when interpreting diary studies!

Clinical Evidence Linking Triggers to Migraine Headaches

Much of the data linking triggers to migraine headache have come from patient diary studies, which are fraught with methodologic weaknesses. Randomized controlled trials and prospective diary studies have been conducted with a number of triggers and provide stronger supporting evidence of an association. Table 10-4 lists the strength

Table 10-4. Strength of Evidence of Migraine Triggers

Trigger Factors	Strength of Evidence*	Migraine Trigger	General Headache Trigger
Stress	Strong	Yes	Yes
Menstruation	Strong	Yes	Yes
Caffeine withdrawal	Strong	Unknown	Yes
Visual stimuli	Strong	Yes	Yes
Weather changes†	Strong	Yes	Yes
Nitrates	Moderate	Yes	Yes
Fasting	Moderate	Probable	Yes
Sleep disturbances	Moderate	Possible	Yes
Wine	Moderate	Yes	Yes
MSG‡	Moderate	Unknown	Yes*
Aspartame	Moderate	Yes	Unknown
Smoking	Weak	Not proven	Not proven
Odor	Weak	Not proven	Not proven
Chocolate	Weak	Not proven	Not proven
Tyramine	Weak	Not proven	Not proven

Adapted from Martin VT. Toward a rational understanding of migraine trigger factors. Med Clin North Am 2001;85:911–41.

MSG = monosodium glutamate.

*The strength of evidence was defined as follows: 1) strong: at least two prospective, randomized controlled, or diary studies confirming an association with no dissenting studies; 2) moderate: at least one randomized controlled trial or a prospective diary study confirming an association with no dissenting studies or two supporting studies with one dissenting study; or 3) two or more dissenting trials on a given trigger or no prospective trials at all.

†Studies of the Chinook winds.

‡Studies of MSG-sensitive patients.

of evidence of the individual migraine triggers and whether they have been proven to be migraine or general headache triggers.

An association between a trigger and migraine headache does not prove a cause-and-effect relationship. Some of the triggers could be part of the migraine prodrome (eg, sleep disturbances, stress, emotions, food cravings) and not causally related to migraine headache. Such a theory, however, would not explain the randomized controlled trials documenting some triggers to be precipitants of headaches (eg, monosodium glutamate, wine, caffeine withdrawal, aspartame). Also, it would not explain the association of triggers to migraine headache that are not part of the migraine prodrome (eg, fasting, menstruation, nitrovasodilators, visual stimuli, weather changes).

Recommendations

Despite great advances in the understanding of the pathophysiology of migraine headache, little is known of how migraine headaches are actually triggered. As demonstrated in this case, patients want to know both the "cause" and the "triggers" of migraine headache. Such knowledge gives patients a sense of control over a disorder that strikes episodically, often without a recognizable initiating event. Identification of triggers may allow patients to prevent some of their headaches and should be part of the treatment armamentarium offered to all patients with migraine headache.

The following recommendations can be given regarding the identification and management of migraine triggers:

- Decide if there is a need for trigger factor modification. Trigger factor intervention may not be worthwhile for those with infrequent or nondisabling headache.
- Trigger factors need to be prioritized for each individual patient. In the author's opinion, behavioral and biologic triggers are more important to modulate than dietary triggers.
- Identification of triggers can occur through self-report by the patient or through the use of a headache diary. Interpretation of a headache diary can be tricky since trigger factors may only provoke a headache 42 to 82% of the time after an exposure.
- Avoidance of triggers should be undertaken for all possible and definite triggers when possible. Some triggers cannot easily be avoided, such as menstruation, stress, depression, and allergies, and specific medications may need to be given to treat these conditions.

Selected Readings

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Editorial Comments

Dr. Martin describes a frequent clinical problem—the patient who mistakes the cause of migraine for a trigger of migraine. He then provides an erudite and thorough review of the literature on triggers and headache, with very clear clinical recommendations for the headache practitioner. He states, and the editors agree, that evaluation of triggers by history and diary must be part of conventional treatment for all headache patients. However, Dr. Martin points out that severe, unreasonable dietary restriction rarely results in sustained clinical improvement.

Final diagnoses:

Episodic migraine without aura, chronic tension-type headache