

How Do I Discuss Hormones with My Migraine Patients?

Common Patient Questions Answered

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1. What are hormonal headaches and menstrual migraine?

Hormonal headaches are headaches that occur in relationship to hormonal changes including menses, post-partum, perimenopause and menopause. These hormonal changes may be occurring “endogenously” from a woman’s own fluctuating hormones (estrogen and progesterone from her ovaries) or “exogenously” from a contraceptive or hormone treatment being used, including birth control pills as well as hormone treatments in the form of patches, pills and gels.

Hormonal migraine, if in relationship to menses, are more appropriately called menstrual migraine. These are disabling migraine headaches that occur by definition within two days prior to the onset of a woman’s menses up to three days into her menses where day one is considered the first day of bleeding. If this pattern occurs at least 66% of the time, it is considered menstrual migraine.

Menstrual migraine is then subdivided into two types: pure menstrual migraine and menstrual-related migraine. Women who experience pure menstrual migraine only have migraine attacks associated with menses. In contrast, women with menstrual-related migraine, the more common subtype, can experience migraine attacks at other times of their cycle from other triggers such as stress, weather change, dehydration, etc.

2. Why do hormonal headaches occur?

The primary reason hormonal headaches occur is a drop or change in brain estradiol concentration. Just before menses, estradiol levels drop, and this is felt to be the most important factor in menstrual migraine. There is also some estradiol fluctuation at ovulation that accounts for an ovulation-related headache. Estradiol levels climb in pregnancy and many women experience marked improvement in their migraine. At delivery, estradiol levels drop precipitously and often cause worsening of migraine attacks during the postpartum period. Breastfeeding can help delay the return of migraine because it can delay resumption of hormonal cycling. During perimenopause, typically ages 47-51, a marked fluctuation of hormones often leads to a time of hormonal headache exacerbation.

If women go into menopause spontaneously, migraine improves in 2/3 of those women. If women undergo abrupt surgical menopause with removal of their ovaries, only 1/3 of women have improvement of their headaches.

3. How are hormonal headaches different from headaches that aren't triggered by hormones?

Hormonal headaches tend to be more disabling and last longer than non-hormonal headaches. For some women, they can last five to seven days and are often more refractory to traditional acute and preventive migraine treatment.

4. Is there anything important to know about the risk factors associated with menstrual migraine (like thrombotic events)?

Women who experience migraine with aura (characterized by reversible neurological signs and symptoms lasting typically from five to 60 minutes prior to headache onset) are at higher risk of stroke and blood clots than women in the general population. There is no significant increased risk in women who have migraine without aura. This is why most health care providers are reluctant to use estrogen-containing contraception in women who have migraine with aura but feel comfortable using estrogens in women with migraine without aura.

Women with migraine can reduce their risk of stroke and blood clots by not smoking, keeping blood pressure under control, being normal body weight, monitoring their cholesterol and seeing their providers regularly.

5. What can someone do if they are experiencing menstrual migraine?

First, it is important to keep a headache diary or calendar (paper or electronic) to confirm the diagnosis. Once the diagnosis is confirmed by a health care provider, migraine-specific acute medications like the "triptans" can be prescribed. They come in oral, injectable and nasal spray formulations. The most common triptan prescribed is sumatriptan. It is generic and affordable for most patients.

Newer migraine acute treatment options have now become available including the "gepants" ubrogepant and rimegepant as well as a 5-HT_{1F} receptor agonist lasmiditan. These newer agents may be an option for women who can't tolerate the triptans or for whom the triptans are ineffective or contraindicated.

A common approach can be short-prevention of menstrual migraine by taking a preventive approach during the vulnerable time of the cycle. For example, a patient can start taking ibuprofen or naproxen several days before the anticipated menstrual migraine and continue until the menses is complete. Alternatively, about 200-250 mg of magnesium taken twice a day can also be used preventively and be started several days prior to the anticipated onset of menstrual migraine. Other treatment strategies can include short-term prevention with one of the triptans, e.g. naratriptan 2.5 mg or frovatriptan 2.5 mg taken 1 once a day or twice a day for five to seven days during a woman's perimenstrual window or what she has identified as the most vulnerable time of her cycle.

For women who have migraine without aura, hormonal approaches can be very helpful, including continuous low-dose oral contraceptives (while skipping the placebo to keep a more even level of estradiol), continuous back-to-back use of the combined vaginal ring or wearing an estradiol patch as "add-back" estrogen to help prevent the drop in estradiol just before menses. Of these options, use of the low-dose vaginal ring provides the most constant, least fluctuating estrogen environment. Although the contraceptive transdermal patches also eliminated hormonal fluctuations, their estrogen exposure is higher than with the currently marketed contraceptive vaginal ring.

Reference

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