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MIGRAINE AND HORMONAL CYCLES: IMPLICATIONS FOR WOMEN'S HEALTH

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ABSTRACT

A common chronic, intermittent primary headache illness that primarily affects women, migraine affects 15% to 18% of adult individuals. In years spent living with a disability, migraine is ranked second. Worldwide and is among the ten most incapacitating conditions, based on data from the 2016 Global Burden of Disease. Women's reproductive systems, in contrast to men's, show regular cyclic modifications that could be teleologically understood as a regular cycle of preparation for fertilization and pregnancy. The period of cyclic ovarian function spans the brief periods of increasing or decreasing ovarian activity between puberty and menopause. Despite being a continuum, the menstrual cycle is commonly shown as starting on the menstrual cycle's first day and concluding on the final day before the subsequent menstrual cycle. The quality of life for migraineurs is enhanced by locating and removing migraine triggers. In a study conducted in Belgium, 217 migraineurs (176 women and 41 males) showed that older women with longer disease duration had a greater incidence of migraine triggers. The possibility of poly-triggers like food and drink was elevated when menstruation was present. According to the research, ovulation (8.5%) was the most frequent cause of migraines, meals (47.7%), menses (48%), stress (48.8%), and alcoholic beverages (51.6%). Postmenopausal women have been examined to examine the impact of estrogen and the usage of HRT on migraines because menopause is a natural condition of estrogen shortage. Woman's menstrual cycle, her estrogen levels change. Furthermore, perimenopause, menopause, lactation, and pregnancy all involve protracted alterations in estrogen levels.

Hormone replacement treatment (HRT) and oral contraception also affect estrogen levels. Although the relationship between hormones and headaches is complex. Twenty to sixty percent of women who report having migraines say they get them during their menstrual cycle. Severe, throbbing headaches, light and sound sensitivity, nausea, and vomiting are common symptoms of migraines. These symptoms can make day-to-day activities difficult and even incapacitated. This paper presents a summary of publications on migraine and female hormone fluctuations from 2010 through 2023 to establish benchmark for migraine and hormonal cycles affect women's health and to support the country's healthcare system to improve community well-being.

KEYWORDS: Migraine, Hormonal Phases, Hormonal changes, Quality of life, Menstrual cycle, Menstrual Migraine, menopause, migraine triggers.

INTRODUCTION

A common chronic, intermittent primary headache illness that primarily affects women, migraine affects 15% to 18% of adult individuals. In years spent living with a disability, migraine puts second worldwide and is between the ten most incapacitating conditions, based on the 2016 Global Burden of Disease statistics. There are two types of migraine: those with an aura and those without.[1] Temporary neurological symptoms, primarily affecting the visual field and frequently lasting 5–60 minutes before the onset of migraine headaches, are what define the aura. Many concomitant illnesses, including cardiovascular disease, are more common in migraineurs, and some research has directly connected this increased risk to the aura subtype. There may be mechanical differences in migraine across the sexes, given the high frequency of the illness in women, which suggests a different contribution from genetic effects.

Variations in migraine severity between the sexes

It has long been known that female sex hormones play a role in migraine promotion at every stage of a woman's life. Beginning after menarche, the increased occurrence of migraine in women peaks at age 40 and declines following menopause. During the first trimester of pregnancy, the perimenopause, and the time of their monthly cycles, many women report having more migraine attacks. A decline in progesterone and estrogen levels is correlated with the commencement of monthly bleeding. A major area of research for studies examining sex differences in migraine has been the influence of gonadal hormones, particularly oestrogen.[2]

Studies have indicated that the frequency of migraine attacks is higher when estrogen levels are declining and lower when estrogen levels are rising. During the late luteal phase, female migraineurs also show quicker estrogen withdrawal than controls. These findings provide credence to the theory that migraine attacks may be brought on by withdrawal of estrogen combined with changes in other hormones, like oxytocin.

For women with migraines, replacing estrogen with contraceptives is a common component of hormonal rehabilitation. Changes in sex hormones have been shown to impact CGRP release and initiate the trigeminovascular system.[3] Women who have regular menstruation tend to have higher levels of CGRP in their tear fluid and plasma. The idea that stable estrogen levels could potentially modify CGRP and protect against migraine is reinforced by the observation that women on estrogen-containing oral contraceptives have CGRP levels that are similar to those of non-migraineurs women.

Reproduction is a phenomenon in nature that requires precise synchrony to produce a successful outcome. Throughout the course of a menstrual cycle, all biological and physiological mechanisms connected fertility as well as the potential for conception in females are tightly managed, preserving an exact hormonal balance.[4]

Menstrual Cycle and Migraine

Women's reproductive systems, in contrast to men's, show regular cyclic modifications that could be teleologically understood as a regular cycle of preparation for fertilization and pregnancy. Recurrent vaginal bleeding, or menstruation, which is the result of uterine mucosa being shed, is the most obvious feature of the menstrual cycle in humans and primates. On average, the menstrual cycle lasts for 28 days, despite the cycle's recognized unpredictability in length. Starting with the first day of menstruation, the days of the cycle are typically numbered. It starts at puberty, which occurs between the ages of 10 and 16, and ends with menopause, which normally occurs at 51.[5]

Period of proliferation or follicles

The period of proliferation or follicles is the menstrual cycle’s initial phase. It occurs between day one and day fourteen of the menstrual cycle, lasting an average of 28 days. Variations the duration of the, the fluctuation in the menstrual cycle duration is due to the follicular phase. Estrogen, or 17-beta-estradiol, is the main hormone throughout this phase. FSH receptors on

the follicle during the cycle's onset are unregulated, which increases this hormone. However, once the follicular phase concludes, because 17-beta-estradiol levels are increased, the anterior pituitary will receive negative input. Uterus's endometrial layer is supposed to expand during this phase. The uterine endometrial layer is stimulated to develop, along with the quantity of stroma and glands, and the spiral arteries supplying the endometrium are deepened by 17-beta-estradiol.[5]

During ovulation

On day 14 of a normal 28-day cycle, ovulation takes place, as it always occurs 14 days prior to menstruation. At the end of the proliferative cycle, follicular maturation and increased hormone synthesis lead to high levels of 17-beta-estradiol. Currently, the only hormones that 17-beta-estradiol favourably promotes are FSH and LH. At least 200 picograms of 17-beta-estradiol per millilitre of plasma must be present for it to reach a critical concentration. Prolonged periods of high FSH and LH are referred to as the LH surge. This causes the mature follicle to split, releasing an oocyte.

Secretory phase

After the menstrual cycles secretory phase, comes the luteal phase. This phase always includes the 14th through 28th day of the cycle. During this phase, progesterone - which is induced by LH - is the main hormone that gets the corpus luteum and the endometrium ready for the potential implantation of fertilized eggs. Progesterone will give the anterior pituitary negative input once the luteal phase ends, lowering the FSH and LH amounts, thus reduces progesterone and 17-beta-estradiol levels. Because of the negative feedback loop, the corpus luteum develops at the mature follicle rupture point in the ovary, an organization that generates progesterone and 17-beta-estradiol, this near the end of the period is plentiful.

Menstrual cycle endocrinology

The ovarian function cycle includes the brief phases of rising or decreasing ovarian activity that occur between adolescence and menopause. The menstrual cycle is a continuum; however, it is commonly represented as starting on the first day of the period and terminating on the last day before to the following one. The shift between ovulation, the organization and collapse of the corpus luteum, and the gradual changes in hormone levels, after follicular expansion with elevated oestrogens, is addressed by this subjective peripheral assessment of withdrawal of steroid hormones. Growth of the next cohort of follicles had already started by the time of the next menstrual cycle.[6]

Gonadotropin-releasing hormone (LHRH or & RI-I) is secreted by the hypothalamus, the pituitary, which releases luteinizing hormone and The hormone called follicle-stimulating hormone (FSH) and glycoproteins, progesterone our estrogen secreted next to the ovary, along with the uterine endometrial lining, responds to estrogen and progesterone, must work in unison for this system to function normally.[7] Under nor epinephrine, serotonin, endogenous opioids, Neurotransmitter control, including corticotropin-releasing hormone (CRH), the hypothalamus secretes GnRH, which promotes pituitary LH and FSH. As a result, ovarian estrogen and progesterone are stimulated. Consequently, the hypothalamus controls GnRH and the pituitary modifies the LH and FSH ratios.

Opioids and CRH limit G&I-I secretion, but nor epinephrine promotes it. Intraneuronal prostaglandin E2 may also directly control t6 GnRH secretion. The secretion of GnRH is pulsatile as opposed to continuous. This plasticity is required because the pituitary is not stimulated by continuous GnRH secretion; also the regularity and amplitude in the pulses affect the result of FSH and LH. Pulses occur every one to two hours throughout the follicular stage of the cycle.[8]

When the corpus luteum secretes progesterone during the luteal phase of the cycle, the frequency of episodic gonadotropin release steadily decreases. Just before menstruation, this process virtually ends. Progesterone secreted by the corpus luteum increases the frequency of episodic gonadotropin release during the luteal phase of the cycle. Just before menstruation, this process nearly entirely ends.[9]

Migraine triggers

The quality of life for migraineurs is enhanced by locating and removing migraine triggers.

In a study conducted in Belgium, 217 migraineurs (176 women and 41 males) showed that older women with longer disease duration had increased occurrence of migraine triggers. The potential for poly-triggers like food and drink was elevated when menstruation was present.[10] According to the study, ovulation was revealed to be the most frequent migraine trigger (8.5%), meals (47.7%), menses (48%), stress (48.8%), and alcoholic beverages (51.6%). This research emphasizes how important stress and hormones are in the development of migraines.[1]

Migraine triggers are

- Stress.

- Dietary triggers.
- Hormonal fluctuations.
- Sleep disturbances.
- Barometric pressure and other weather changes.
- Sensory stimulation.
- Drug overuse.
- Dehydration.
- Skipping meals regularly.
- Alcohol.

Stress ranked first among the 15 categories in most of the included research (90%) and was sleeping patterns and eating/food habits come next (83 and 79%, respectively). Conversely, however, the categories of medication (2%), alcohol (24%), travel (24%), and allergy/sinus (2%), were the least studied. Furthermore, stress was identified as the primary trigger in 66% of the research papers, while sleep was listed as one of the top three triggers in 74% of them.[11] In general, migraine triggers that were asked about more frequently (such as stress and sleep) had a higher prevalence than those that were asked about less frequently (like vacations and alcoholic beverages). However, A few were an exception, like hormones, which despite greater awareness shown a lower frequency as triggers.[7]

The Role of Estrogen and Progesterone

Postmenopausal women have been examined to examine the impact of estrogen and the usage of HRT on migraines because menopause is a natural condition of estrogen shortage. The first proof that estrogens cause headaches dates back to 1972, when Somerville showed that treating female migraine patients intramuscularly with pro-drug ester of 17 β-estradiol [E2] (estradiol valerate), but not progesterone, right before menstruation, postponed the onset of menstrual migraine. Following an exogenous estrogen priming period, the levels of circulating E2 (45–50 pg/mL) found to be the threshold below which migraines were particularly induced. After the intramuscular injection of E2, postmenopausal women on HRT were studied to confirm a threshold for estrogenic activity.[12] These results suggested that the menstrual migraine attacks might be caused by an estrogenic withdrawal effect. Nowadays, aside from the correlation between a drop in estrogen and migraine onset after prior exposure to estrogen, it is likely that physiologic oscillations in estrogen also play a role in migraine etiology. In a comparison between the patients' late luteal phase estrogen decline

rate and that of healthy controls, it was clear that female migraineurs had a unique neuroendocrine sensitivity.[9]

Throughout a woman's menstrual cycle, her estrogen levels change. Furthermore, perimenopause, menopause, lactation, and pregnancy all involve protracted alterations in estrogen levels. Hormone replacement treatment (HRT) and oral contraception also affect estrogen levels. Although the relationship between hormones and headaches is complex, it's vital to think about whether this element may be influencing your migraines. It's unclear exactly how variations in estrogen trigger migraines, but there are a few potential explanations.[13] It is well recognized that estrogen affects serotonin's function, a neurotransmitter involved in mood and pain regulation. Blood vessels and blood pressure are influenced by estrogen, and blood vessel changes are known to contribute to migraines. These two elements, along with possibly other ones, could operate as mediators in the relationship between estrogen and migraines.[13]

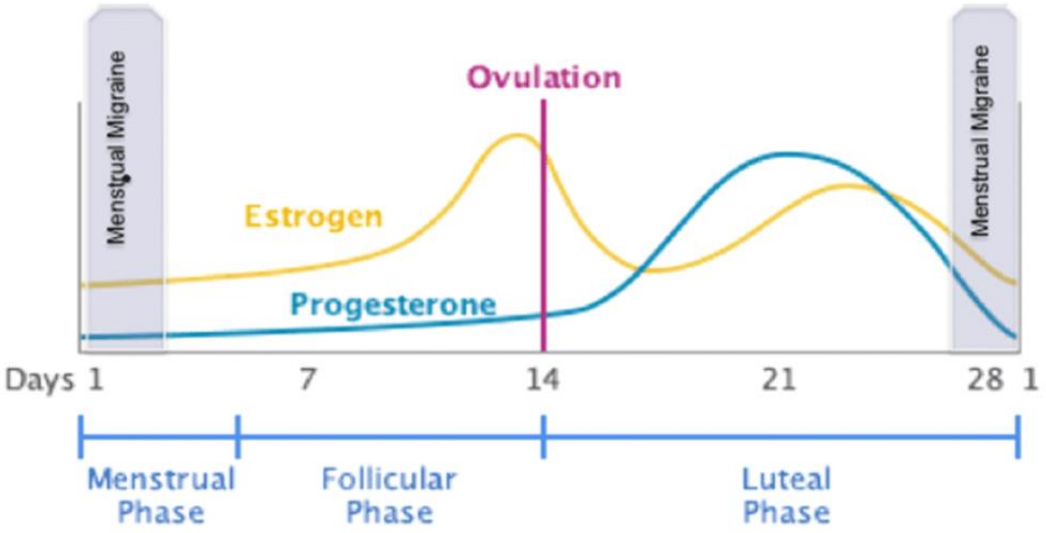

Figure 1: Showing the complex relationship between menstrual Cycle and Menstrual migraine. (Cupini, L. M *et al*., 2020)

Menstrual migraine

Twenty to sixty percent of women who report having migraines say they get them during their menstrual cycle.[15] A migraine that happens during the perimenstrual stage, which starts two days before the onset of menstrual flow and ends around day three of a woman's cycle, is

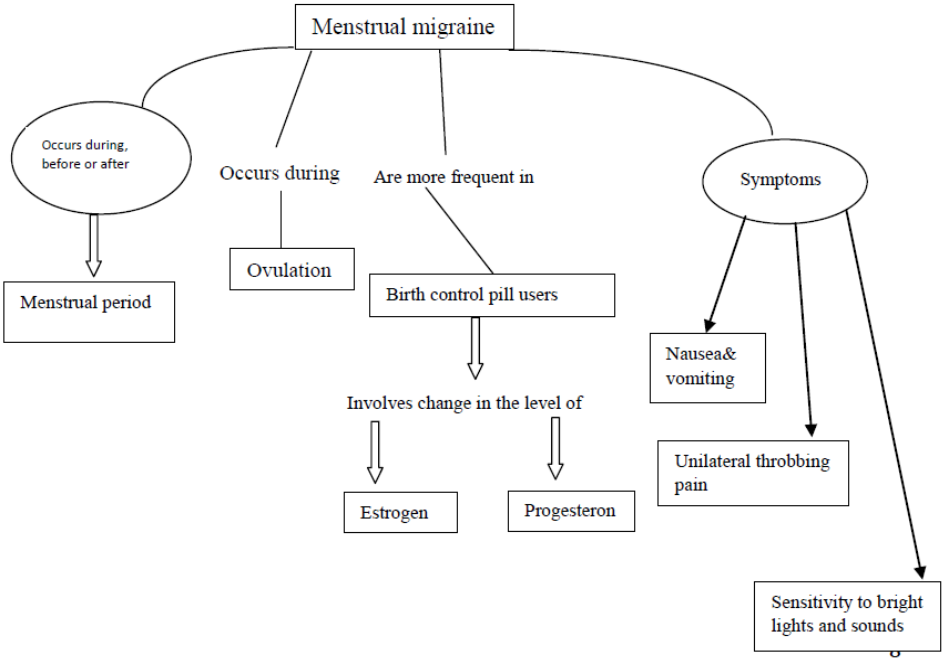

Figure 2: Effect of estrogen and progesterone in menstrual migraine.

Known as a menstrual migraine. Estrogen levels decrease throughout this time. A woman's menstrual cycle has two dips in estrogen: one immediately following ovulation, which occurs two weeks after the end of the menstrual cycle, and another before the onset of the menstrual cycle. While they are less common and typically less severe than menstrual migraines, some women also get migraines throughout their menstrual cycles.[4]

Management and Treatment strategies for migraine

Identifying environmental factors: A variety of environmental elements have been found and documented to be linked to the start of a migraine attack. Certain people with migraine are triggered by changes in the weather, such as low air pressure, excessive humidity, and low temperatures. The relationship between these stimuli and migraines is associated with pathophysiological mechanisms that are poorly understood.[16] However, it's hypothesized that neuropeptides like substance P or CGRP are released in response to certain triggers, such as low barometric pressure. In addition to the atmospheric pressure fluctuations that can result in migraine episodes, additional travel-related factors that might induce headaches include inadequate air conditioning, uncomfortable seating, dietary changes, and sleep problems. Similarly, a variety of causes, such as hypoxia, exposure to cold temperatures, intense physical exercise, irregular sleep patterns, and dietary issues, are linked to climbing.[17] While raising altitude and lowering oxygen levels might cause migraine headache attacks, it's important to keep in mind that, The ICHD-III guidelines state that

"headache attributed to airplane travel" is considered a distinct illness and should be taken into account at all times.[18]

Sleep modification: Disruptions to sleep are among the most common causes of migraines An increased frequency of attacks is associated with sleep disorders like insomnia and shifts in the amount of time spent sleeping overall (either too little or too much), Inappropriate timing and low-quality sleep, which includes difficulty falling asleep, early morning alertness, and disturbed sleep. Furthermore, it's commonly recognized that shifting the timing and length of sleep on the weekends or during holidays might cause migraine attacks. However, for certain migraineurs, sleep is proven to be a component in headache alleviation. There is a bidirectional relationship between headaches and sleep disturbances, meaning that headaches can induce sleep disturbances and sleep issues can cause headaches.[17]

Dietary modification: As was previously noted, nutrition plays a key role in migraine management. As a result, diet factors and how they cause migraine attacks should be taken into account. Slow-digesting foods are those that maintain a stable blood glucose level; prepare meals rather than grabbing fast food or processed foods; drinking enough water to stay hydrated; eating frequently; avoiding certain food items (listed below); eating a low-fat diet; and so on. Dietary lifestyle modification can involve a variety of strategies. The gut-brain axis, which describes the connections between the gut and brain, is not well understood. But inflammatory variables, the gastrointestinal tract's microbial makeup, neuropeptides, the serotonin pathway, and Bodily hormones related to stress can affect how the brain and intestines are associated.[18]

Chocolate, red wine, alcohol, and foods high in caffeine are a few popular foods that trigger migraines (tea, coffee, cola, chocolate, and a few medications), foods high in nitrates, ice cream, processed meals, and some seafood (ham, salami, hot dogs, and bacon), tyramine-rich foods (Bananas, avocados, citrus fruit, aged cheese, beans, and non-fresh meat), as well as meals rich in monosodium glutamate.[18]

Impact on quality of life

Physical health: Severe, throbbing headaches, light and sound sensitivity, nausea, and vomiting are common symptoms of migraines. These symptoms can make day-to-day activities difficult and even incapacitated.[19]

Mental health: Anxiety, depression, and other mood problems are more common in people who suffer from chronic migraines. Migraine's unpredictable nature and high intensity can cause severe psychological suffering.[20]

Work and Productivity: Frequent absences from work and decreased productivity are two consequences of migraines. Ineffective task execution might hinder career advancement and job happiness.[7]

Social life: Due to their ability to restrict social connections and activities, migraines can cause relationship tension and isolation. Social interactions may be further limited by the need to avoid triggers (such as particular meals, lights, or stressful situations).

Cognitive function: People may have trouble focusing, memory issues, and disorientation during and after a migraine episode. These symptoms can interfere with day-to-day activities and decision-making.[21]

Sleep: Migraines may cause an increase in The regularity and intensity of their assaults disruption of sleep patterns, which can result in insomnia or poor-quality sleep.[22]

Financial impact: The price of prescription drugs, medical appointments, and the possible loss of revenue from missed work days can all add up to a big financial burden.

Quality of life: The constraints imposed by migraines and chronic pain frequently result in a lower overall quality of life. Once delightful activities could become difficult or unfeasible. The overall quality of life is frequently diminished as a result of migraine restrictions and chronic pain. Once fun activities may now be difficult or unattainable.[23]

Future directions

Hormonal Therapy and Migraine management: Examine the effectiveness and safety of various hormonal therapies, such as hormone replacement therapy and contraceptives, in the treatment of menstrual migraines. Examine individualized hormonal treatment programs based on migraine patterns and individual hormonal profiles.

Research on Genetic and Biomarkers: Find biomarkers and genetic markers that indicate a person's propensity for hormonal migraines. Examine how heredity affects how differently women suffer migraines.

Effects of menstrual phases: To gain a better understanding of how the menstrual cycles various phases affect migraine frequency and severity, conduct longitudinal studies. Examine non-pharmacological treatments for menstrual migraines, including food adjustments, physical activity, and stress reduction methods.

Pregnancy and the Postpartum period: Investigate how hormonal changes associated with pregnancy affect migraine patterns and Establish guidelines for treating migraines during pregnancy and breastfeeding. Examine how pregnancy affects migraine patterns over the long term in women who have experienced migraines in the past.

Menopause and Migraine: Examine the effects of various menopausal phases, such as perimenopause and post menopause, on migraine symptoms. Examine the advantages and hormone replacement therapy drawbacks for Menopausal migraine therapy.

Psychological and Social effects: Research how women's mental health and quality of life are affected psychologically by hormonal migraines. Examine the effects of hormonal migraines on family life and work productivity, as well as the social and economic ramifications.

Patient Education and Support: Create educational initiatives to give women the knowledge and skills they need to properly manage their migraines by educating them about the connection between hormones and the disease. Establish support networks for women who get hormonal migraines, such as support groups and online forums.

Integration of digital health technologies: To provide individualized insights and treatment regimens, and track migraine patterns and hormone cycles using mobile apps and digital health technologies. Examine remote monitoring and telemedicine options for the treatment of hormonal migraines, particularly in underprivileged areas.

Collaborative approaches: To create all-encompassing migraine therapy plans, encourage collaborative research combining neurologists, endocrinologists, gynecologists, and mental health specialists.

CONCLUSION

The purpose of this document is to enhance the nation's healthcare system and set a baseline for how migraines and women's hormone cycles affect health by summarizing papers on the

subject from 2010 to 2023. This review covers the migraine factors, triggers, hormonal imbalance, effects of estrogen and progesterone, and quality of life which may influenced by migraine in quickly changing cultures. Every year in India migraine affects 15% to 18% of adult individuals.

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