Changes in maternal sleep during pregnancy and pregnancy outcomes

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ABSTRACT

Sleep disorders in pregnancy are incompletely studied, as they are significant health problems with maternal-fetal implications. These are quite common due to the hormonal, anatomical, and functional changes that occur in the mother’s body. Sleep deprivation influences the mother’s health, with important repercussions on the fetus. Polysomnography shows that the changes regarding sleep architecture begin in the first trimester, and disturbances are also observed after birth. Obstetrical implications (way of birth, duration of labor, analgesia, anesthesia at birth, early onset of labor) and maternal conditions (hypertension induced by pregnancy, gestational diabetes, mental disorders) can change sleep quality. Early identification of sleep disorders, as well as prompt prenatal management, especially through non-pharmacological means, is essential to avoid negative consequences.

INTRODUCTION

During pregnancy, sleep disorders frequently occur due to hormonal secretion, adaptive functional and anatomical changes, and fetal movements [1]. Hormonal changes during pregnancy are the most important triggers of sleep disorders. Thus, the increased secretion of estrogen and progesterone affects the duration and quality of sleep. In the first trimester of pregnancy, the increased secretion of progesterone by inducing GABA2 receptors leads to excessive sleepiness [1,2].

During pregnancy, secondary to the increase in estrogen, there is vascular congestion and hyperemia, which causes edema of the mucous membrane of the upper respiratory tract. In addition, progesterone causes a dilation of the upper airways by increasing the activity of the genioglossus muscle, thus having a protective effect against obstructive sleep apnea (OSA). However, there is an increased risk of secondary central apnea with chemoreceptor reset and hyperventilation-hypocapnia predominance [1,3]. During pregnancy, in addition to the increase in steroid hormones, there is an increase in prolactin, melatonin, oxytocin, and cortisol, disturbing the diurnal rhythm [2].

Among the anatomical changes in pregnancy that cause sleep disturbances to be mentioned is weight gain in pregnancy by more than 20% compared to pre-gestational weight, as well as increased uterine volume. Respiratory failure can also occur by affecting the elevation of the diaphragm secondary to the increase in uterine volume, which leads in the last trimester of pregnancy to a 20% decrease in residual functional capacity [1,4].

The functional adaptive changes of the pregnant woman’s body are also involved in the occurrence of sleep disorders. Thus, cardiorespiratory changes such as increased heart rate, blood pressure, cardiac output, and respiratory rate lead to maternal fatigue and exhaustion. Physiological digestive changes contribute to sleep disturbances, such as increased gastric emptying time with slower digestion, constipation, and gastroesophageal reflux. Urinary changes such as frequent nocturnal urination can also affect sleep [1,5]. Sleep can also be disturbed due to fatigue, nausea, vomiting, and body pain [5,6].

Also, body positioning difficulties can affect both the initiation and continuity of sleep, adding to back pains and leg cramps. Repeated sleep loss leads to an allostatic load with unfavorable results in pregnancy [1].

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During pregnancy, sleep is modified, and common sleep-related complaints are difficulty initiating sleep, shorter total sleep duration, more time awake after falling asleep, more time in light sleep stages, and less rapid eye movement (REM) or deep sleep [7,8].

Pregnant women have more frequent multiple sleep disorders than the general population. Pregnant women who are overweight and over 30 years of age have more frequent sleep disorders and are at increased risk of complications. Depending on the time of onset of sleep disorders in pregnancy, they can have different side effects. Thus, their appearance in the first two trimesters increases the risk of gestational diabetes, and in the third trimester, the risk of hypertensive disorders [9], longer labor, and cesarean delivery [7].

Breathing disorders associated with sleep disorders during pregnancy increase the risk of hypertensive pathology through hypoxia, negatively affecting the mother and fetus. Thus, it is important to report them and perform polysomnography for the diagnosis [10].

In this article, we wanted to highlight the main sleep disorders that occur during pregnancy and the importance of identifying and treating them to significantly reduce the risk of fetal and maternal injury.

SLEEP DISTURBANCES DURING PREGNANCY

Poor sleep quality

Poor sleep quality, most commonly defined by decreased sleep efficiency and deep sleep, is assessed by the Pittsburgh Sleep Quality Index (PSQI) with a value ≥ 5. It is found in approximately 29% to 76%, with an average of 45.7% of pregnant women, and occurs more frequently towards the end of the third trimester [6,11].

The earlier the sleep quality is affected in pregnancy, the more the risk of complications increases. Poor sleep quality causes gestational hypertension and gestational diabetes [1,12].

It leads to increased systemic inflammation with the occurrence of premature birth [6] and small for gestational age infants [13,14]. Also, studies have shown a 20% increase in prolonged labor and cesarean delivery [15]. In addition, poor sleep quality is a potential trigger for both prenatal and postpartum depression and anxiety [6,13].

Short or long sleep duration

The average sleep duration in pregnant women compared to non-pregnant ones is lower by 0.5 hours. In the first trimester of pregnancy, the night sleep time is 8.8 hours, falls to 8.3 hours in the middle of pregnancy, and remains constant at 8.4 hours until birth [14,16].

Changes in sleep duration refer to either shortening it to less than 7 hours a night or extending it to more than 9 hours [9]. The short sleep duration varies from 5.6% in the first trimester of pregnancy up to 9.6% in the third one [14]. Sleep deprivation increases the risk of labor duration, gestational diabetes, cesarean section rates [17,18,19], premature birth, and small for gestational age infants [14]. Disturbances in sleep duration, both by shortening and lengthening it, influence glucose metabolism and insulin production. Synergistically prolonged sleep duration, overweight, or obesity before pregnancy causes gestational diabetes [20,21].

Insomnia

Insomnia is a sleep disorder characterized by difficulty falling asleep or staying asleep, which occurs at least 3 times a week and can be diagnosed using the Insomnia Symptom Questionnaire (ISQ) [22]. It is found in 38.2% of pregnant women, most frequently in the third trimester of pregnancy, due to stress and increased physical demand compared to the first part of the pregnancy. In addition, a pregnant woman over 20 years increases the risk of insomnia [23,24]. Both, depression and anxiety in early pregnancy are associated with greater symptoms of insomnia and sleepiness in later stages of pregnancy [25].

Secondary to insomnia, pregnant women experience fatigue, hypersomnia, and mood swings throughout the day [7]. Insomnia increases the rate of premature births, large for gestational age newborns and cesarean sections [26,27], as well as postpartum depression [1].

Restless legs syndrome

Restless legs syndrome (RLS), also known as Willis-Ekbom disease, is a neurologic sensorimotor disorder that occurs in 22-30% of pregnant women [28,29]. The appearance of RLS in pregnancy is associated with an increase in steroid hormones, especially estradiol, which reduce the sensitivity to dopamine in the nigrostriatal. The increase in thyroid hormones during pregnancy and iron deficiency limits the conversion of tyrosine into dopamine, also contributing to the occurrence of RLS. Prevalence increases with maternal age and parity, being stationary after the third pregnancy. Other predictive factors for gestational RLS to be mentioned are low ferritin levels, positive family history, snoring, nocturnal leg cramps, and excessive daytime sleepiness. In symptomatic women before pregnancy, symptoms are exacerbated during pregnancy [1,30].

The diagnosis is clinical and consists of the presence of an impulse to move the legs, which can be as-
Sociated with unpleasant and disturbing sensations during periods of rest, predominantly nocturnal, which improve with movement. The biological evaluation of iron, ferritin, and transferrin levels is also useful [30]. These symptoms negatively affect pregnant women, causing sleep disorders and stress. They also affect intellectual, physical, and occupational activities and social relationships. The relationship is bidirectional, RLS can be aggravated by pathologies associated with pregnancy, such as gestational diabetes, preeclampsia, or postpartum depression. The symptoms get worse as the pregnancy progresses, they go away in the last month of pregnancy and at birth and are maintained during the postpartum period [1, 30]. In women who had RLS during pregnancy, the risk of chronicity of the disease increases four times [31].

More frequent episodes of RLS during pregnancy increase the probability of gestational hypertension, small-for-gestational-age, and low-birth-weight newborns [32].

Sleep-disordered breathing

Sleep-disordered breathing (SDB) encompasses changes in breathing from snoring to complete cessation of breathing (apnea). Apnea is the interruption of air flow with a minimum duration of 10 seconds and is accompanied by oxygen desaturation and, thus fragmented sleep. Hypopnea is the interruption of airflow for more than 10 seconds, with more than 50% reduction in airflow and 3% desaturation. Thus, the severity of SDB is usually expressed in terms of apnea and hypopnea index (AHI) ≥5, depending on the number of events that occur during one hour of sleep [1, 33]. Obese pregnant women have a higher AHI than non-obese pregnant women [34].

SDB may occur secondary to increased diaphragmatic effort leading to suction pressure in the upper airway, ultimately increasing upper airway collapsibility. Also, SDB can result from rhinitis and nasopharyngeal edema caused by increased estrogen. So there is a low permeability of the upper airways and increased resistance to airflow [1, 35]. Snoring occurs in 14-45% of pregnant women, especially in the third trimester [36].

SDB leads to sleep fragmentation and thus increased sympathetic activation, with the resistance of the upper airways and the appearance of arterial hypertension. Preeclampsia can result from endothelial dysfunction produced by hypoxia, oxidative damage, or inflammation with secondary cytokine activation. In addition, changes in angiogenesis markers and glycoproteins secreted by the placenta are observed [1, 36]. SDB favors adult metabolic dysfunctions, especially in males [1].

Snoring is also a predictive factor for small gestational age fetuses and gestational diabetes [1, 37]. Snoring through intermittent hypoxia and sympathetic overload of the placental circulation increases fetal erythropoiesis, with an increase in red blood cells, as well as erythropoietin and interleukin-6 in the vessels of the umbilical cord [1].

Obstructive sleep apnea

Obstructive sleep apnea (OSA) is a disorder that occurs during pregnancy in 15% of women [38]. OSA has an increased prevalence in pregnant women with higher BMI. The objective diagnosis is made by polysomnography assisted overnight [34].

In pregnant women with OSA, there is sympathetic stimulation, recurrent hypoxemia, alteration of glucose tolerance and insulin resistance, and chronic systemic and vascular inflammation with negative effects on both the mother and the fetus [2]. Thus, OSA can cause the early onset of gestational hypertension, preeclampsia, gestational diabetes, and premature birth. OSA increases the duration of labor, as well as the rate of cesarean sections. Also, increases the risk of premature birth, stillbirth, small for gestational age, or large for gestational age. Due to prolonged labor, hypoxic fetal brain lesions may occur, with lower Apgar scores at birth and neonatal intensive care [1, 3, 39, 40].

SLEEP DISTURBANCES AND MATERNAL OUTCOME

Hypertensive disorders in pregnancy

Sleep deprivation or its fragmentation causes hypertensive disorders, such as pregnancy-induced hypertension or preeclampsia, secondary to intermittent hypoxia, oxidative stress, inflammatory responses, and vascular or neural mechanisms. Thus, vascular damage causes endothelial damage, atherosclerosis, or even thrombosis, with changes in the diameter of small vessels and cardiac output, increasing blood pressure, as well as activation of the hypothalamic-pituitary axis or the renin-angiotensin-aldosterone system. Neuronal mechanisms involve an increase in central sympathetic activity, with a decrease in heart rate and variable blood pressure, by affecting the baroreflex function [9, 41].

Gestational diabetes mellitus

Sleep disorders, except insomnia, increase the risk of developing gestational diabetes. This can be explained by circadian desynchronization secondary to sleep fragmentation or short duration, with endocrine changes such as increased insulin resistance and diabetes due to abnormal sympathetic activation [9]. Along with sleep disorders, the occurrence of gestational diabetes is favored by the hypothalamic-pituitary axis, endothelial dysfunction, oxidative
stress, increased inflammation, and immune dysfunction [1,2].

SLEEP DISTURBANCES AND FETAL OUTCOME

As with hypertensive disorders, placental or umbilical cord lesions or thrombosis can cause placental dysfunction and hypoperfusion, with fetal growth dysfunction, premature birth, or fetal death [9].

Preterm birth

Sleep disorders such as insomnia, OSA, and S-SSB are associated with premature birth in 30-40% of cases [42]. The short duration and poor sleep quality, especially in the third trimester, increase the risk of premature birth twice [14].

Premature birth occurs secondary to inflammation caused by lack of sleep, with increasing inflammatory cytokines such as interleukin-6 (IL-6) and IL-8. They stimulate the production of prostaglandin and thus cause uterine contractions [9,14]. Another explanation of the appearance of premature birth may be related to the disruption of the adrenal-hypothalamus-pituitary axis that is adjusted by sleep. Thus, sleep disorders increase the cortisol level, which plays a role in the development and maturation of fetal organs. The increased cortisol level can cause a domino effect, with modification of the progesterone/estrogen hormone balance by lowering progesterone and thus causing birth. In addition, besides sleep disorders, high levels of stress in the third trimester of pregnancy lead to increased maternal cortisol [14,43].

Small for gestational age

Fetal growth can also be influenced by sleep disorders. Small for gestational age newborns (SGA) have a birth weight < 10th percentile for gestational age. The increase of the inflammation secondary to the low sleep duration determines the decrease of the bioavailable nitric oxide, which ensures maternal-fetal exchanges, growth, and fetal development [14,44]. Also, as a result of sleep disorders, insulin resistance can occur in pregnant women, influencing fetal weight at birth [14]. In the case of severe snoring during pregnancy, the risk of low birth weight increases 3 times [45].

Large for gestational age

Large for gestational age (LGA) fetuses have a birth weight >90th percentile for gestational age. LGA is another fetal consequence that can result from sleep disorders, especially in the case of insomnia [9].

Nocturnal hypoxemia is associated with the severity of LGA, especially in the case of advanced maternal age or comorbidities such as chronic hypertension, obesity, and pregestational diabetes [46].

The chances of an LGA fetus are 5-6 times higher in the case of pregnant women with OSA in the third trimester of pregnancy [40]; on the contrary, some studies do not prove this [47,48]. OSA in pregnancy is associated with increased placental weight and excessive expression of placental leptin, which may cause increased adiposity in the newborn [49].

Stillbirth

Fetal death can occur due to maternal hypotension secondary to prolonged maternal sleep, to which a sedentary lifestyle, low body temperature, and low arousal also contribute [9]. In the last month of pregnancy, OSA, sleep duration > 9 hours, and daytime sleep increase the chances of stillbirth [50,51]. In addition, the mother's sleeping position in the supine position can increase the risk of stillbirth, especially in the case of prolonged sleep and lack of awakening [51,52].

TREATMENT OF SLEEP DISTURBANCES DURING PREGNANCY

Early and accurate diagnosis and treatment of sleep disorders in pregnancy are essential to reduce complications.

Non-pharmacological treatment

Treatment is initially non-pharmacological for most sleep disorders during pregnancy and breastfeeding. Fortunately, most sleep problems during pregnancy improve or remit once the baby is born.

Sleep hygiene is essential, creating a comfortable sleeping environment with low ambient lights at night and choosing an appropriate bedtime and wake-up time. It is important to avoid consuming food, many liquids, alcohol, caffeine, nicotine, or sedatives before sleep, as well as watching TV or using the phone [1,7].

Position during sleep is an important factor influencing sleep quality and pregnancy outcomes. In recommended to sleep on the left side, with knees and hips bent, with one leg over the other, and with a pillow between them [7].

Daily moderate intensity physical exercises, especially those performed in water and relaxation, of at least 30 minutes a day, 4-6 hours before bedtime, improve the quality of sleep [7,53,54]. Yoga, meditation, acupuncture, massage, and cognitive-behavioral therapy are useful in the management of sleep disorders, especially insomnia [9,55].

For the treatment of SDB, OSA, and insomnia, continuous positive nasal pressure (CPAP) and standard prenatal care are recommended in cases refractory to other methods [1,9]. Oral devices are also used for...
OSA [9]. CPAP ventilation therapy for pregnant women with OSA and severe forms of SDB can reduce nocturnal blood pressure [56]. In addition, the beneficial effects of CPAP have also been proven on insulin secretion in pregnant women with gestational diabetes [57].

**Pharmacological treatment**

In the case of RLS, folate and iron supplements are administered orally (ferritin < 75 mcg/L) or intravenously (ferritin < 30 mcg/L). Clonazepam in low doses (0.25-1 mg/day) or gabapentin (300-900 mg) can be administered in the evening, in the last two trimesters of pregnancy, and during breastfeeding in case of refractory RLS. It is recommended to avoid the combination of anticonvulsants. Also, levodopa or carbidopa in a dose of 50-200 mg can be administered to prevent the worsening of RLS. Opiates, despite their effectiveness on RLS, must be administered with caution because they can cause congenital heart disease or neonatal withdrawal syndrome in exposed infants. In the case of the coexistence of maternal heart disease or neonatal withdrawal syndrome, benzodiazepines, lorazepam, must be done with caution and at the minimum effective dose, as there is a risk of fetal palate defects, fetal sedation, or respiratory disorders. Antidepressants, anxiolytics, and hypnotics can be administered from the beginning of pregnancy in the case of symptomatic women to prevent the occurrence of severe sleep disorders without the risk of congenital malformations [2,7,58].

SDB usually responds very well to non-pharmacological methods, but in some cases, when comorbidities such as rhinitis or asthma are associated, specific pharmacological treatment is necessary. Rarely, in OSA, in addition to CPAP, the administration of antimuscarinic drugs is necessary [58].

**CONCLUSIONS**

Sleep disorders in pregnancy have negative maternal and fetal effects. Early diagnosis and treatment are important to reduce complications. Standard gynecological care should consider implementing effective programs regarding sleep disorders.

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