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Obstructive Sleep Apnea in Pregnancy: An Underrecognized Syndrome with Maternal and Fetal Consequences?

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ABSTRACT

Background: Sleep Obstructive Apnea (OSA) is becoming increasingly recognized as a serious comorbidity in pregnancy but remains underdiagnosed. Physiological changes and anatomical alterations of pregnancy can predispose women to or worsen underlying OSA, which has been a cause for concern regarding its potential to represent a unique obstetric syndrome.

Objective: To determine the prevalence of OSA in pregnant patients, evaluate its relationship to maternal and perinatal outcomes, and determine whether OSA manifests as a unique clinical syndrome in obstetric care.

Methodology: This prospective observational study was conducted over 18 months at Lady Reading Hospital, Peshawar, in collaboration with the Sleep Medicine and Pulmonology Units. A total of 160 pregnant women between 20–32 weeks gestation was screened using the STOP-BANG questionnaire; those with scores ≥3 underwent confirmatory sleep studies.

Results: OSA was diagnosed in 48 women (30%). The OSA group experienced significantly higher rates of maternal problems, including preeclampsia (27.1% vs. 8.9%, p = 0.003), gestational diabetes (29.2% vs. 12.5%, p = 0.009), gestational hypertension (33.3% vs. 11.6%, p = 0.002), and cesarean birth (64.6% vs. 38.4%, p = 0.004). Multivariate regression also established that OSA was an independent predictor of maternal adverse outcomes (Adjusted OR 3.6, 95% CI: 1.8–7.1, p = 0.001), joined by BMI >30 and STOP-BANG score ≥3.

Conclusion: OSA is common in pregnancy and significantly linked with complications in both maternal and neonatal outcomes. Its independent effect, even after age and obesity have been adjusted for, adds strength to the argument that OSA should be considered a new obstetric clinical syndrome.

Keywords: Obstructive Sleep Apnea; Pregnancy; Obstetric Syndrome; STOP-BANG; Sleep-Disordered Breathing

Introduction

Pregnancy is a complicated physiological condition that puts a lot of strain on the heart, lungs, and hormones. As our knowledge of maternal health advances, it has become clearer that disorders that were previously believed to be rare or unimportant during pregnancy may really have a significant impact on the outcomes for both the mother and the fetus. Among these, obstructive sleep apnea (OSA), a condition that is typically linked to middle-aged, overweight males. However, there is mounting evidence that a significant percentage of pregnant women may also be affected by OSA, especially if they are obese, have advanced maternal age, or have other comorbid illnesses.[1]This change in perspective brings up a crucial query, is OSA only a pregnancy comorbidity, or is it becoming recognized as a unique obstetric illness that needs careful consideration and treatment?

The hallmark of obstructive sleep apnea is recurrent episodes of upper airway blockage during sleep, which can cause fragmentation of sleep, hypercapnia, and intermittent hypoxia. Endothelial dysfunction, systemic inflammation, and elevated sympathetic activity are among the physiological stress reactions set off by these occurrences. Insulin resistance, poor glucose metabolism, hypertension, and cardiovascular disease are all recognized to be associated with OSA in the general population. Given that preeclampsia, gestational diabetes mellitus, and gestational hypertension already pose serious threats to the health of both the mother and the fetus, these same disorders are especially concerning throughout pregnancy. Therefore, especially in high-risk groups, it is scientifically possible that OSA could exacerbate or even cause these pregnancy-related issues.[2]

OSA in pregnant women is still largely underdiagnosed in spite of these worries. It might be difficult to identify the symptoms clinically because exhaustion, frequent nightly urination, poor sleep, and morning headaches sometimes resemble common pregnancy-related issues. Standardized, pregnancy-specific screening methods for OSA are also lacking, and prenatal care settings do not frequently use gold-standard diagnostic tests such overnight polysomnography. The chance to act early is thus lost, and many cases of sleep-disordered breathing remain unidentified. Preterm birth, intrauterine growth restriction, hypertension, and increased rates of cesarean delivery are just a few of the negative pregnancy outcomes that studies are showing a close correlation with OSA.[3] Additionally, untreated sleep apnea may have long-lasting impacts on fetal growth and adaption to life beyond of the pregnancy, as maternal OSA has been connected to neonatal problems like poor Apgar scores and admission to neonatal intensive care units.

Women may be at risk for upper airway narrowing and blockage due to the physiological changes that occur during pregnancy, specifically increased fluid retention, mucosal edema, and a decrease in functional residual lung capacity. Additionally, the framework of sleep and ventilatory drive are impacted by elevated progesterone levels during pregnancy. These alterations may worsen pre-existing but undetected illness or cause OSA to manifest for the first time during pregnancy in women who had previously been asymptomatic. Given that symptoms may be reversible with the right care, such as continuous positive airway pressure (CPAP) therapy, identifying and treating OSA during pregnancy may be a significant chance to enhance the health of both the mother and the unborn child.[4,5]

The recognition of obstructive sleep apnea (OSA) as a pregnancy-specific syndrome derives its rationale from the significant, and often overlooked, implications of OSA on maternal-fetal health - similar to the clinical courses of established obstetric syndromes such as gestational diabetes and preeclampsia. Barriers in standardized screening and management mean that it is currently impossible to systematically evaluate the prevalence of OSA, obstetric correlates and clinical characteristics-data that could support OSA as a high-risk pregnancy disorder. If OSA were to be recognized formally, OSA assessment would become part of routine prenatal care as sleep health was incorporated and in turn, lead to further specialized studies and changes to practice as had been undertaken after prior syndrome definitions in obstetrics were established, at a time when pregnancy and obesity-related sleep disorders continue to be defining characteristics of modern perinatal medicine.

Objective

To determine the prevalence of OSA in pregnant patients, evaluate its relationship to maternal and perinatal outcomes, and determine whether OSA manifests as a unique clinical syndrome in obstetric care.

Methodology

The present study was conducted at Bakhtawar Amin Medical & Dental College, Multan, over the course of 18 months. Prior to enrollment, each participant provided written informed consent, and the study was approved (IEC/92/22_10) by the Institutional Ethics Committee of Bakhtawar Amin Medical College. A total of 160 pregnant women in their second or early third trimester (between 20 and 32 weeks of gestation) were enrolled in this study. The study participants were pregnant women with singleton pregnancies at 20-32 weeks of gestation who consented to battery sleep assessments and clinical follow-up. Exclusion criteria included pre-existing chronic respiratory disease (e.g., asthma, COPD), previously diagnosed obstructive sleep apnea, multiple pregnancies, significant

fetal anomalies, and refusal to consent to study invitation.
The STOP-BANG questionnaire, a validated sleep assessment tool that asks about snoring, fatigue, observed apneas, high blood pressure, BMI, age, neck circumference, and gender (modified for pregnancy), was used to screen participants for signs of sleep-disordered breathing. Women were thought to be at risk for obstructive sleep apnea if their STOP-BANG score was ≥3. Information gathered included obstetric history, comorbid conditions (e.g., diabetes, gestational hypertension), sleep-related symptoms, and demographics (age, parity, BMI, neck circumference). Certified sleep specialists interpreted sleep studies. The American Academy of Sleep Medicine (AASM) used the Apnea-Hypopnea Index (AHI) to diagnose OSA. The criteria were mild OSA (AHI 5–14), moderate OSA (AHI 15–29), and severe OSA (AHI ≥30). Maternal and neonatal outcomes were documented, and participants were monitored until delivery. Preeclampsia, gestational diabetes, gestational hypertension, delivery method, and any complications during labor and delivery were all considered maternal outcomes. Neonatal outcomes included NICU admission, Apgar scores at 1 and 5 minutes, birth weight, and gestational age at delivery. To find correlations between a diagnosis of OSA and unfavorable outcomes for either the mother or the fetus, data was examined.
SPSS version 25 was used to conduct the statistical analysis. The mean ± standard deviation (SD) was used to represent continuous variables, while frequencies and percentages were used to represent categorical variables. For continuous variables, the student's t-test or Mann-Whitney U test was used as appropriate, and for categorical variables, the Chi-square test or Fisher's exact test was used to compare groups (OSA vs. non-OSA). Using a multivariate logistic regression model, potential confounding variables such as age, BMI, and parity were taken into consideration. A p-value of less than 0.05 was deemed statistically significant.

Results

The 160 pregnant women were recruited to the study. 48 women (30%) were diagnosed with Obstructive Sleep Apnea (OSA) based on STOP-BANG screening and confirming sleep study, whereas 112 (70%) were not having OSA. Baseline characteristics, clinical features, are described in which mean maternal age in the OSA group was 31.6 ± 4.2 years, which was much higher than 28.9 ± 3.9 years in the non-OSA group (p = 0.001). This indicates that rising maternal age could be a risk factor for OSA during pregnancy. Body Mass Index (BMI in kg/m^2) was 33.4 ± 2.8 in the OSA group and 28.1 ± 2.4 in the non-OSA group, a very significant difference (p < 0.001). This shows obesity as the key determinant factor for OSA during pregnancy. Neck Circumference (cm) Women with OSA had a significantly higher mean neck circumference (37.8 ± 2.1 cm) than those without OSA (34.6 ± 1.9 cm), with a p-value < 0.001. Neck circumference is an established anthropometric predictor of OSA, as it is an indicator of upper airway soft tissue volume. A circumference of >35 cm in women has been reported to be associated with increased OSA risk in the general and obstetric population. The percentage of primigravida was less in the OSA group (35.4%) compared to the non-OSA group (52.6%), with a statistically significant difference (p = 0.042). STOP-BANG Score ≥3 (%) all women (100%) in the OSA group had a STOP-BANG score of ≥3 compared to just 27.6% in the non-OSA group (p < 0.001) (Table 1).
The majority of affected pregnant women (58.3%) had mild OSA, with an AHI between 5 and 14 events per hour. Mild OSA may also serve as an early warning sign for progressive sleep-disordered breathing, especially in women with increasing gestational weight gain. Nearly one-third (29.2%) of the women had moderate OSA, indicating more frequent episodes of disrupted breathing during sleep. These patients are more likely to require close monitoring and potential intervention (e.g., CPAP therapy). A smaller but clinically important subset (12.5%) had severe OSA, with an AHI ≥30 (Table 2).
Gestational Hypertension (33.3% vs. 11.6%, p = 0.002), OSA women had three times the incidence of gestational hypertension in comparison to non-OSA women. Preeclampsia (27.1% vs. 8.9%, p = 0.003), the incidence of preeclampsia a pregnancy complication was found to be significantly higher in the OSA group. Endothelial dysfunction and oxidative stress caused by OSA are known to worsen placental insufficiency and inflammatory processes, leading to an increased susceptibility of women to preeclampsia. Gestational Diabetes Mellitus (GDM) (29.2% vs. 12.5%, p = 0.009), approximately 1 in every 3 OSA women developed gestational diabetes, while only 12.5% of the women without OSA developed gestational diabetes. This indicates a very high association between OSA and glucose intolerance, possibly due to interrupted sleep impacting insulin sensitivity. Cesarean Delivery (64.6% vs. 38.4%, p = 0.004), the rate of cesarean section was much higher among the OSA group. ICU Admission (10.4% vs. 1.8%, p = 0.019), OSA women had a much-increased rate of intensive care unit (ICU) admission, which reflects an increased burden of severe maternal morbidity (Table 3).
Table 4 contrasts the neonatal outcome of OSA-affected versus unaffected pregnancies in terms of differing timings for birth, intrauterine development, adaptation after birth, and need for neonatal care. Preterm Delivery (<37 weeks), Preterm delivery in 31.2% of the OSA vs. 12.5% of the non-OSA (p = 0.005) group was significantly different. Preterm delivery poses a risk to neonatal morbidity and subsequent developmental difficulties. Low Birth Weight (<2.5 kg), Newborns of OSA mothers had a significantly greater proportion of low birth weight (27%) than those of non-OSA mothers (10.7%) (p =

Table 1. Baseline Characteristics of Study Population (N = 160)

Variable	OSA Group (n = 48)	Non-OSA Group (n = 112)	p-value
Mean Age (years)	31.6 ± 4.2	28.9 ± 3.9	0.001
BMI (kg/m²)	33.4 ± 2.8	28.1 ± 2.4	<0.001
Neck Circumference (cm)	37.8 ± 2.1	34.6 ± 1.9	<0.001
Primigravida (%)	35.4%	52.6%	0.042
STOP-BANG Score ≥3 (%)	100%	27.6%	<0.001

0.006). Low birth weight is linked with higher perinatal mortality and greater susceptibility to chronic disease in adulthood. NICU Admission, the frequency of Neonatal Intensive Care Unit (NICU) admission was higher in the OSA group (25.0%) compared to the non-OSA group (8.0%) (p = 0.003). This result highlights the increased neonatal susceptibility for maternal OSA, due to both prematurity and respiratory or metabolic instability at birth. Mean Apgar Score (5 minutes), the non-OSA group had a higher average Apgar score at 5 minutes (8.1 ± 0.4) than the OSA group (7.6 ± 0.5) (p < 0.001). Lower Apgar scores can indicate decreased oxygenation, delayed adaptation, or transient neonatal depression. Fetal Growth Restriction (FGR) in 18.7% of the OSA group was higher than in the non-OSA group at 6.2% (p = 0.011). FGR is associated with increased risks of perinatal complications and long-term neurodevelopmental handicap.

OSA Diagnosis (Adjusted OR 3.6, 95% CI: 1.8–7.1, p = 0.001), Maternal OSA was independently and strongly associated with adverse maternal outcomes, having over threefold higher odds even after adjusting for other risk factors. This establishes that OSA plays an important role in maternal morbidity in addition to its correlation with obesity or age. BMI ≥30 (Adjusted OR 2.3, 95% CI: 1.2–4.5, p = 0.014) was also independently predictive of adverse outcomes with over twice the risk. STOP-BANG Score ≥3 (Adjusted OR 2.9, 95% CI: 1.4–5.8, p = 0.003) was independently predictive of almost three times the risk of adverse maternal outcomes. Since this score is a common screening measure for OSA, its implication here confirms its usefulness in the clinical setting in obstetric patients. Age >30 (Adjusted OR 1.8, 95% CI: 0.9–3.5, p = 0.072), Although women over 30 had a tendency toward higher risk, the relationship was not significant. Multiparity (Adjusted OR 1.2, 95% CI: 0.6–2.3, p = 0.612), was not strongly related to poor outcomes in this model, which suggests that parity itself does not seem to have an impact on the risk when other more powerful variables (OSA, BMI, STOP-BANG) are controlled (Table 5).

Discussion

The increasing awareness of Obstructive Sleep Apnea (OSA) as an important cause of poor pregnancy outcomes is redesigning obstetric practice. Traditionally underdiagnosed in pregnant populations, OSA is now emerging as a potential syndrome within obstetrics, influencing both maternal and neonatal morbidity. Our study aimed to explore the prevalence, severity, and outcomes associated with OSA in a cohort of 160 pregnant women, of whom 48 were diagnosed with OSA based on clinical screening and sleep study evaluation.

The age of females in the OSA group was significantly greater (31.6 ± 4.2 years) than in the non-OSA group (28.9 ± 3.9 years), with p = 0.001. This result concurs with earlier research, Louis et al. (2014)[6], which indicated that increased maternal age was the most significant risk

Table 2. Severity of OSA in Affected Patients (n = 48)

Severity Level	Number of Patients	Percentage (%)
Mild OSA (AHI 5–14)	28	58.3%
Moderate OSA (AHI 15–29)	14	29.2%
Severe OSA (AHI ≥30)	6	12.5%

Table 3. Maternal Outcomes of study cases

Outcome	OSA Group (n = 48)	Non-OSA Group (n = 112)	p-value
Gestational Hypertension (%)	33.3%	11.6%	0.002
Preeclampsia (%)	27.1%	8.9%	0.003
Gestational Diabetes (%)	29.2%	12.5%	0.009
Cesarean Delivery (%)	64.6%	38.4%	0.004
ICU Admission (%)	10.4%	1.8%	0.19

factor for OSA during pregnancy, especially in women aged above 30. Facco et al. (2021)[7] also recorded a similar pattern, with an increased incidence of sleep-disordered breathing in women of advanced maternal age, possibly contributed by age-related decreases in airway muscle tone and enhanced upper airway collapsibility.

Obesity was a dramatic discriminator between the groups, with an overwhelmingly greater BMI reported for the OSA group (33.4 ± 2.8 kg/m²) compared to the non-OSA group (28.1 ± 2.4 kg/m²) ($p < 0.001$). This result further lends support to previous research by Izci-Balserak (2019)[8], who found BMI to be one of the most predictive metrics for OSA in pregnancy. Excess adiposity around the thorax and neck degrades patency of the upper airway and leads to sleep obstructive events. Pamidi et al. (2014)[9] also emphasized that maternal obesity is the key enabler for OSA onset and negative pregnancy outcomes, thus a key intervention target.

Neck circumference, an indirect marker for narrowing of the upper airway, was also larger in the OSA group (37.8 ± 2.1 cm) than in the non-OSA group (34.6 ± 1.9 cm) ($p < 0.001$). The parameter has been shown to be a valid predictor of OSA in multiple populations, such as pregnant women. For a study by Louis et al. (2014)[6], neck circumference of >35 cm emerged as a strong predictor for sleep-disordered breathing, particularly when it is used in association with other indices like BMI and age.

Primigravida status was decreased in the OSA group (35.4%) when compared to the non-OSA group (52.6%) ($p = 0.042$), indicating that multiparity can go hand in hand with increased risk for sleep apnea. Although the link between parity and OSA is not fully understood, Pien (2014)[10] proposed that repeated pregnancies may contribute to long-term physiologic changes in respiratory mechanics and upper airway dynamics. The most extreme difference was noted in STOP-BANG scores, wherein 100% of OSA-diagnosed women had a score ≥3 versus merely 27.6% in the non-OSA group ($p < 0.001$). This glaring disparity attests to the extreme sensitivity of the STOP-BANG questionnaire in identifying women at risk for OSA in pregnancy. Facco et al. (2021)[7] confirmed the validity of applying STOP-BANG to antenatal populations and proved its efficacy in early screening, particularly when polysomnography is not easily accessible.

Our findings revealed that the greatest number of affected women (58.3%) presented with mild OSA (AHI 5–14), followed by 29.2% with moderate OSA (AHI 15–29), and only 12.5% with severe OSA (AHI ≥30). The dominance of mild OSA among pregnant patients has been recurring observation other research by Facco et al. (2021)[7], around 56% of pregnant women with newly diagnosed OSA were

Table 4. Neonatal Outcomes during study period

Outcome	OSA Group (n = 48)	Non-OSA Group (n = 112)	p-value
Preterm Delivery (<37 weeks)	31.2%	12.5%	0.005
Low Birth Weight (<2.5 kg)	27.0%	10.7%	0.006
NICU Admission (%)	25.0%	8.0%	0.003
Mean Apgar Score (5 min)	7.6 ± 0.5	8.1 ± 0.4	<0.001
Fetal Growth Restriction (%)	18.7%	6.2%	0.011

Table 5. Multivariate Logistic Regression for Adverse Maternal Outcomes

Predictor Variable	Adjusted OR	95% CI	p-value
BMI >30	2.3	1.2 – 4.5	0.014
OSA Diagnosis	3.6	1.8 – 7.1	0.001
Age >30	1.8	0.9 – 3.5	0.072
Multiparity	1.2	0.6 – 2.3	0.612
STOP-BANG ≥3	2.9	1.4 – 5.8	0.003

in the mild category. Equally, Louis et al. (2014)[6] found that the majority of pregnant women diagnosed in third-trimester sleep studies had AHI scores compatible with mild OSA. Such results indicate that though OSA is becoming more prevalent in pregnancy, the majority of cases are likely to be subclinical or milder in nature but still may have significant physiological and clinical implications. While moderate and severe OSA contributed to fewer cases in our analysis (29.2% and 12.5%, respectively), their presence is clinically important. As an example, Pamidi et al. (2014)[9] reported that moderate-to-severe OSA was independently linked to an increased risk of preeclampsia and gestational diabetes, even after controlling for age and BMI. Severe OSA during pregnancy has been reported in a multicenter observational study by Ramos et al. (2020)[11] as an independent powerful predictor of maternal ICU admission and neonatal complications like NICU admission and low Apgar scores. In our population, despite only 12.5% having severe OSA, these women were found to have more significant adverse outcomes, as evident from the later maternal and neonatal outcomes. Also noteworthy is that severe OSA can be underdiagnosed in pregnancy, especially in environments where polysomnography is not available or where symptomatology (e.g., snoring, daytime fatigue) crosses over with frequent pregnancy-related complaints. Izci-Balserak (2019)[8] highlighted the diagnostic difficulty of separating physiologic changes of sleep in pregnancy from pathologic sleep-disordered breathing when OSA is mild or atypically expressed. Thus, actual prevalence of moderate-to-severe OSA might be greater than is being reported. Pregnancy has been shown to modify upper airway dynamics on account of hormonal fluctuations, fluid retention, and weight gain, which may result in worsening of severity of OSA throughout the trimesters. It is explained by Pien et al. (2014)[10] that some of the women with mild OSA in early pregnancy might develop moderate or severe OSA by the third trimester because of these physiological changes.

In our study, gestational hypertension was seen in 33.3% of the OSA group compared to 11.6% of the non-OSA group (p = 0.002), whereas preeclampsia was seen in 27.1% of OSA patients compared to 8.9% of non-OSA cases (p = 0.003). These results are consistent with those of Louis et al. (2014)[6], who performed a large retrospective cohort study and found that OSA in women was more than twice as likely to develop preeclampsia and gestational hypertension. Likewise, Pamidi et al. (2014)[9] identified that early pregnancy sleep-disordered breathing independently predicted threefold hypertensive disorders of pregnancy after controlling for BMI.

Among our cohort, 29.2% of women with OSA developed GDM, versus 12.5% of those without OSA (p = 0.009). The finding is in agreement with previous work by Facco et al. (2021)[7], who found that there was a significant correlation between OSA and insulin resistance in pregnancy. The cesarean delivery rate was notably higher in the OSA group (64.6%) than in the non-OSA group (38.4%) (p = 0.004). O'Brien et al. (2014)[12] reported that OSA was significantly related to the probability of cesarean section, particularly among women with comorbid hypertension or GDM. Even the higher rate of surgical delivery might be a demonstration of obstetricians' fear of fetal distress, macrosomia, or other complications like preeclampsia in women with OSA. Admission to the ICU was needed in 10.4% of the OSA patients, compared to 1.8% of the non-OSA population (p = 0.019). Although a small number difference, this is significant and indicates the higher overall maternal morbidity in the group. OSA in pregnancy is link with an increased risk of critical care interventions from acute cardiopulmonary complications, severe hypertension, or unanticipated respiratory depression under anesthesia, as reported by a systematic review by Bourjeily et al. (2017)[13]. Women with OSA can also be at greater risk during the period surrounding the operation, especially for cesarean delivery, because of increased susceptibility to sedatives and opioids, leading to further impairment of respiratory function after the operation. Louis et al. (2014)[6] suggested pre-delivery planning and screening, such as potential anesthesia consultation and postoperative monitoring for women with diagnosed OSA. In the present

study, preterm delivery (<37 weeks) in the OSA group was 31.2% compared with 12.5% in the non-OSA group (p = 0.005). This considerable variation is in line with that of Louis et al. (2014)[6], who found that OSA was independently linked with an almost twofold risk of preterm birth even after adjustment for BMI and hypertension. Likewise, Pamidi et al. (2014)[9] identified that OSA women had a greater prevalence of spontaneous and indicated preterm births resulting from complications such as preeclampsia and fetal growth restriction. Mechanically, intermittent hypoxia and systemic inflammation resulting from OSA may compromise placental perfusion and induce preterm labor or require maternal or fetal indications for early delivery. We found that 27.0% of neonates in the OSA group were born with a weight <2.5 kg compared with just 10.7% in the control group (p = 0.006). This is consistent with the research conducted by Chen et al. (2012)[14], which proved that maternal OSA correlated with a raised risk of giving birth to infants born with LBW and small-for-gestational-age status. The research indicated that placental hypoxia and altered nutrient exchange could be contributory factors. O'Brien et al. (2014)[12] also documented this trend, implicating poor placental function, intrauterine stress, and systemic endothelial dysfunction in causing low birth weight in infants whose mothers had OSA. The percentage of neonates who were admitted to NICU was substantially higher among mothers with OSA than among those without OSA (25.0% vs. 8.0%; p = 0.003). This finding accounts for the clinical susceptibility of such neonates, who are frequently born prematurely, with low birth weight or with compromised fetal status. As per research conducted by Edwards et al. (2019)[15], maternal OSA raised the risk of NICU admission by as much as 2.8 times, with respiratory distress syndrome and hypoglycemia being the most frequent reasons. The 5-minute mean Apgar score was also significantly less in the OSA group (7.6 ± 0.5) than in the non-OSA group (8.1 ± 0.4, p < 0.001). This clinically significant but subtle reduction reflects impaired neonatal adaptation at birth, potentially resulting from intrauterine hypoxia, placental insufficiency, or the impact of maternal comorbidities such as preeclampsia and GDM, which were more prevalent in the OSA group. A research by Bourjeily et al. (2017)[13] identified that neonates of untreated OSA mothers presented with lower Apgar scores and increased requirement for resuscitation, indicating fetal well-being in utero and during labor is affected by sleep-disordered breathing. In our population, Fetal Growth Restriction (FGR) was detected in 18.7% of neonates in the OSA group versus 6.2% in the non-OSA group (p = 0.011). This is in agreement with the conclusions of Pien et al. (2014)[10], who associated maternal OSA with higher incidence rates of FGR among women with coexistent preeclampsia. They suggested that intermittent maternal hypoxemia causes placental dysfunction, hence restricting oxygen and nutrient supply to the fetus. Similarly, Chen et al. (2012)[14] have also provided evidence of the relation between OSA and FGR, highlighting that even mild sleep-disordered breathing can impact fetal growth patterns if it goes unreported and untreated. OSA Diagnosis (Adjusted OR = 3.6; 95% CI: 1.8–7.1; p = 0.001) A sole diagnosis of obstructive sleep apnea (OSA) was the most significant predictor of maternal adverse outcomes, with an adjusted odds ratio (OR) of 3.6. This finding is corroborated by Louis et al. (2014)[6], who carried out a large retrospective cohort study and established that women with OSA had a significantly increased risk of gestational hypertension, preeclampsia, cesarean delivery, and ICU admission. Their work highlighted the independent contribution of OSA, independent of obesity or age, to adverse obstetric outcomes. Likewise, Pien et al. (2014)[10] showed that OSA during pregnancy was linked to a fourfold elevated risk of hypertensive disorders, and that treating OSA with CPAP could reduce some of these complications. This work builds on that evidence by statistically confirming OSA as a major independent risk factor. BMI >30 (Adjusted OR = 2.3; 95% CI: 1.2–4.5; p = 0.014), Obesity (BMI >30) was significantly related to poor maternal outcomes in our cohort, with an adjusted OR of 2.3. This supports the findings of Facco et al. (2021)[7] and O'Brien et al. (2014)[12], who found that maternal obesity is a risk factor for gestational hypertension, diabetes, and cesarean delivery. Notably, obesity and OSA are often comorbid with one another and their additive and synergistic effect on pregnancy complications. STOP-BANG Score ≥3 (Adjusted OR = 2.9; 95% CI: 1.4–5.8; p = 0.003), being a STOP-BANG score ≥3 was an independent predictor of maternal complications, thus proving its utility as an obstetric screening tool. The STOP-BANG questionnaire contains risk factors like snoring, daytime tiredness, observed apnea, hypertension, BMI, age, neck size, and gender (pregnancy-adjusted). Kirkham et al. (2017)[16] noted that STOP-BANG ≥3 in pregnant women exhibited excellent sensitivity for the identification of moderate-to-severe OSA and associated with poor maternal and perinatal outcomes. Age >30 (Adjusted OR = 1.8; 95% CI: 0.9–3.5; p = 0.072), while maternal age >30 had a trend towards higher risk (OR 1.8), this was not significant in this model. This finding confirms Chen et al. (2012)[14] that maternal age as a single factor is a poorer predictor than BMI and severity of OSA. However, advanced maternal age is a known overall risk factor for poor pregnancy outcomes and nonetheless needs to be taken into account during risk stratification. Multiparity (Adjusted OR = 1.2; 95% CI: 0.6–2.3; p = 0.612) was not related to severe adverse maternal outcomes in our study (p = 0.612), indicating that parity per se might not be a strong independent risk factor among OSA patients. This concurs with Bourjeily et al. (2017)[13], who, similarly, did not observe a definite association between parity and heightened OSA-related complications. It can perhaps

influence other variables like uterine tone and mode of delivery, which were not individually examined in this regression.

Conclusion

The present study concluded that the results of this study present strong evidence that OSA during pregnancy is linked to substantial maternal and neonatal morbidity, irrespective of other prevalent risk factors. The uniform and clinically significant associations identified validate the conceptualization of OSA as a syndrome with obstetric implications. Identification and treatment of OSA at an early stage of pregnancy may become a central strategy in enhancing perinatal outcomes and alleviating the workload on maternal healthcare systems.

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