

The effects of pregnancy on nasal physiology

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Background: Nasal congestion that is not present before pregnancy represents a distinct clinical entity called pregnancy rhinitis. The aim of this study is to evaluate the clinical characteristics of nasal physiology over the course of pregnancy.

Methods: The study was conducted with 85 pregnant women and 26 nonpregnant controls. We measured nasal airway patency objectively via acoustic rhinometry (ARM) and anterior rhinomanometry (RMM) and subjectively via the Nasal Obstruction Symptom Evaluation (NOSE) scale in each trimester and compared the results to those of the controls.

Results: The NOSE scores of control and pregnant women showed no difference ($p = 0.866$). Minimal cross-sectional area (MCA1; minimal cross sectional area at nasal valve and MCA2; minimal cross sectional area at the level where the head of inferior turbinate is placed) decreased significantly between the first and third trimesters: first trimester 0.37 cm^2 , third trimester 0.31 cm^2 . There was no difference between each trimester with regard to total nasal resistance.

The correlation analysis between the NOSE score and both total volume and MCA1 in all patients showed no significance ($r = -0.10$, $p = 0.318$; $r = -0.04$, $p = 0.654$, respectively).

Conclusion: Pregnancy affects nasal physiology adversely and impairs nasal breathing in some women. However, based on the findings of this study, we concluded that this clinical entity may not be considered as a disease without complementary symptoms despite the presence of objective changes in nasal parameters. © 2014 ARS-AAOA, LLC.

Key Words:

pregnancy; rhinitis; acoustic rhinometry; rhinomanometry; nasal symptoms

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Pregnancy is a condition that affects various organ systems (eg, genitourinary, cardiovascular, endocrine and respiratory) either through increased circulatory placental hormones or the physiological adjustments and adaptations occurring throughout the body of pregnant women.¹ Nasal congestion that is not present before pregnancy represents a distinct clinical entity known as “pregnancy-related rhinitis.” This condition has been defined by Ellegård and Karlson as “nasal congestion present in the last 6 or more

weeks of pregnancy without other signs of respiratory tract infection and with no known allergic cause, disappearing completely within 2 weeks after delivery.”² In previous studies, this condition was reported to affect up to 20% to 40% of women in all trimesters of pregnancy.³⁻⁶ It is a common belief of the public and medical doctors that this condition severely reduces quality of life in pregnant women.⁷⁻⁹ Nasal obstruction during pregnancy has been suggested to cause snoring and obstructive sleep apnea (OSA). OSA may negatively influence the growth of the fetus and lead to gestational hypertension in pregnant women.^{8,10,11} In addition, coexisting asthma may also be exacerbated in these patients.¹²

There are many proposed theories to explain the association between nasal physiological changes and pregnancy; these include effects of sex hormones via enhanced expression of H1 receptors on nasal epithelial cells,¹³ shift of increased plasma volume to extracellular space,¹⁴ the influence of estrogen via increased gland secretion, and vasodilation via both vasoactive intestinal peptide and cholinergic action.^{5,12,15} Recently, nasal mucosal changes during pregnancy have also been attributed to increased

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levels of placental growth factor.^{16,17} Placental growth factor was suggested to induce overgrowth of nasal mucosa, which eventually leads to obstruction of the nasal airway.¹⁸ The mechanisms by which these hormones act on the nasal mucosa and cause nasal physiological changes are still under investigation. Smoking and previous atopy were the only identifiable risk factors in the pathogenesis of nasal congestion during pregnancy.^{19,20} However, there is still considerable controversy regarding the diagnosis, classification, etiopathogenesis, and treatment of this clinical entity.^{20,21}

The aim of this cohort study is to evaluate the clinical characteristics of nasal physiology with regard to trimesters by measuring nasal airway patency both objectively via acoustic rhinometry (ARM) and anterior rhinomanometry (RMM) and subjectively via the Nasal Obstruction Symptom Evaluation (NOSE) scale.

Patients and methods

This cross-sectional observational study was conducted at the University of Uludag, Departments of Otolaryngology and Obstetrics and Gynecology, between June 2013 and August 2013. Low-risk pregnant patients who attended the obstetric outpatient clinic were included in the study. Exclusion criteria were as follows: (1) <18 years of age; (2) history of previous nasal surgery; (3) presence of nasal pathology necessitating surgery such as severe septal deviation, nasal polyposis, or chronic sinusitis; (4) topical steroid or decongestant treatment during past 3 weeks; (5) history of allergic rhinitis and asthma; (6) twin pregnancy; and (7) hypertensive disease of pregnancy. Overall, 85 pregnant women in all 3 trimesters of pregnancy who did not meet the above exclusion criteria were enrolled. These patients were classified into 3 groups according to gestational age, as first (T1), second (T2), and third (T3) trimester. Twenty-six patients in the control group were selected from healthy nonpregnant women of similar age with no nasal pathology, allergic rhinitis, or previous nasal surgery. The study was approved by the ethics committee of Uludag University Medical School and a signed informed consent form was obtained from all patients.

We used the NOSE scale for assessment of nasal obstruction. This questionnaire involves 5 items related to nasal complaints and their effects on quality of life in adults. These items include: nasal congestion, nasal obstruction, trouble breathing through the nose, trouble sleeping, and inability to get enough air through nose during exercise.²² The patients were asked to score each symptom on a Likert scale ranging from 0 to 4 points, in which a higher score indicated more severe complaints. The instrument was then scaled to a total score of 0 to 100 by multiplying the raw score by 5.^{22,23} Because Turkish translation and validation of Turkish translation of the NOSE evaluation has not yet been completed, we used the English version and simultaneous translation of the questions to patients by the same author (O.O.U.). The subjects were examined using

a light source and nasal speculum to rule out any nasal pathology that could interfere with objective tests. Subsequently, the patients underwent measurement of nasal airway by objective tests including ARM and RMM. ARM was performed using a RhinoScan/Rhinometrics® SRE2000 device (Lynge, Denmark). No decongestants were used before the test and the measurements were repeated until 3 optimal curves were observed. The patients were asked to hold their breath after the nasal adaptor was placed at the end of the nostril. Each nasal passage was tested separately. The difference in cross-sectional area (Minimal cross sectional area at nasal valve [MCA1] and minimal cross sectional area at the level where the head of inferior turbinate is placed [MCA2]) and nasal volume from 2 to 5 cm were considered as the main objective outcomes. To overcome the possible effect of nasal cycle on measurements, we added the right mean MCA1 to the left mean MCA1 to get the arithmetic mean of these 2 values. We performed a similar calculation for MCA2. Another objective test was RMM, which measured nasal airway resistance in both expiration and inspiration in Pa/cm³, using the RhinoStream v.2.1, RhinoMetrics® SRE2000 device (Denmark). In this test, patients were asked to breathe through 1 nostril while the other nostril was occluded with a pressure probe. The total resistance was then analyzed using the following formula:

$$\begin{aligned} 1/\text{total resistance} &= 1/\text{right resistance} \\ &+ 1/\text{left resistance during inspiration.} \end{aligned}$$

After all the data from objective assessments were collected, we compared MCA1, MCA2, total volume, and total nasal resistance in each trimester. These results were also compared to the nonpregnant control group. In addition, the median NOSE scores of patients in each trimester and the control group were compared to identify any relationship between gestational age and subjective symptom score.

Statistical analysis was carried out using SPSS 20.0 and Medcalc v12.3.0. Continuous and discrete variables are presented as median (minimum-maximum) and n (%). The Mann-Whitney and Kruskal-Wallis tests were performed for group comparisons. Bonferroni correction was applied for pairwise comparisons. Receiver operating characteristic (ROC) curve analysis was performed to establish cutoff values associated with the measurements. Correlation analysis was performed to examine relationships between variables, and a Spearman correlation coefficient was computed. A value of $p < 0.05$ was set as the statistical significance threshold. A power analysis was conducted to determine the number of participants needed in the present study. The minimum sample size required for each group was determined to be at least $n = 25$ (desired statistical power: 0.80).

Results

This study included 85 pregnant women who were divided into 3 groups according to trimester of pregnancy as T1 ($n = 25$), T2 ($n = 30$), and T3 ($n = 30$) and nonpregnant

controls ($n = 26$). Participants were homogenous in terms of age and there was no difference between patient groups ($p = 0.889$).

The median NOSE scores of pregnant women and nonpregnant controls were 5 (0 to 80) and 10 (0 to 30), respectively. There was no statistical difference between the NOSE scores in each trimester and controls ($p = 0.966$). However, just a few of the nonpregnant women (3/26, 11.5%) had a symptom score higher than 20, in contrast to pregnant women, who had a higher proportion of symptom scores over 20 (27/85, 31.7%). The total volume of the nasal cavity between 2 and 5 cm in nonpregnant controls was 8.70 cm³ (3.96 to 22.74) and there was a nonsignificant decrement in total volume at later stages of gestation ($p = 0.163$). Total volume decreased to 7.21 cm³ (1.90 to 19.20) in T2 and it was 7.29 cm³ (1.50 to 29.01) in T3 (Table 1). The MCA1 and MCA2 values significantly decreased each trimester ($p = 0.003$ and $p = 0.001$, respectively). There was also a statistically significant difference between the nonpregnant control group and pregnant women for both MCA1 and MCA2 ($p < 0.001$ as shown in Table 2). RMM showed a nonsignificant trend of change in total nasal resistance when comparing trimesters ($p = 0.071$). We found that total resistance was 5.35 Pa/cm³ (1.99 to 18.30) in T1 and decreased to 4.15 Pa/cm³ (1.90 to 10.96) in T2. However, resistance increased again to 5.20 Pa/cm³ (1.90 to 12.29) in third trimester (Fig. 1).

We classified patients into two groups as “asymptomatic” and “symptomatic” based on the NOSE score and tried to find cutoff values for total volume, MCA1, and MCA2. However, there was no significance in ROC

analysis, indicating the absence of a cutoff value for positivity of symptom. This finding indicated that to measure nasal volume or cross sectional area does not help us in differentiating symptomatic pregnant women from asymptomatic ones, as shown in Table 3. We performed further analyses to find a correlation between the NOSE score and both the total volume and the cross-sectional area in all patients. There was no relationship between NOSE score and total volume ($r = -0.10$; $p = 0.318$) nor between NOSE score and MCA1 ($r = -0.04$; $p = 0.654$) and MCA2 ($r = -0.02$; $p = 0.799$).

Discussion

During gestation there is a sustained release of female sex hormones (mainly estrogens and progesterone) and placental growth hormone, which have various systemic effects on several organs. The nasal mucosa of women contains several estrogen receptors, which make it susceptible to hormonal changes.²⁴ These hormones may have indirect effects such as vasodilation, increase of blood and extracellular plasma volume, and mucosal thickening.^{13,14,16}

The effects of changes in hormone levels on nasal physiology and on quality of life in women have been evaluated in many studies.^{7,8,18,25–28} In a study by Philpott et al.²⁷ 10 women underwent anterior rhinoscopy, peak inspiratory nasal flow, acoustic rhinometry, anterior rhinomanometry, mucociliary clearance time, and a rhinitis questionnaire in order to assess nasal physiological alterations during the menstrual cycle. The authors suggested that the decrease in nasal volume and significant increase in nasal

TABLE 1. The comparison between each trimester and nonpregnant controls with regard to clinical findings*

Patient groups	Age (years)	Total volume (cm ³)	MCA1 (cm ²)	MCA2 (cm ²)	NOSE
T1 ($n = 25$)	29 (22–38)	8.89 (2.97–14.70)	0.37 (0.06–0.65)	0.37 (0.10–0.93)	5 (0–70)
T2 ($n = 30$)	28.5 (19–38)	7.21 (1.90–19.20)	0.35 (0.10–0.56)	0.36 (0.07–0.87)	5 (0–70)
T3 ($n = 30$)	28 (22–39)	7.29 (1.50–29.01)	0.31 (0.06–0.69)	0.31 (0.04–1.91)	5 (0–80)
Nonpregnant ($n = 26$)	25.5 (17–56)	8.70 (3.96–22.74)	0.40 (0.11–0.82)	0.46 (0.06–0.97)	10 (0–30)
<i>p</i>	0.899	0.163	0.003	0.001	0.966

*Values are median (minimum–maximum). Bold *p* values are significant.

MCA1 = minimal cross sectional area; MCA2 = minimal cross sectional area at the level where the head of inferior turbinate is placed; NOSE = Nasal Obstruction Symptom Evaluation; T1 = first trimester; T2 = second trimester; T3 = third trimester.

TABLE 2. The comparison between pregnant women and nonpregnant controls with regard to clinical findings*

Patient groups	Total volume (cm ³)	MCA1 (cm ²)	MCA2 (cm ²)	NOSE
Pregnant ($n = 85$)	7.74 (1.50–29.01)	0.35 (0.60–0.69)	0.34 (0.04–1.91)	5 (0–80)
Nonpregnant ($n = 26$)	8.70 (3.96–22.74)	0.40 (0.11–0.82)	0.46 (0.06–0.97)	10 (0–30)
<i>p</i>	0.095	<0.001	<0.001	0.866

*Values are median (minimum–maximum). Bold *p* values are significant.

MCA1 = minimal cross sectional area; MCA2 = minimal cross sectional area at the level where the head of inferior turbinate is placed; NOSE = Nasal Obstruction Symptom Evaluation.

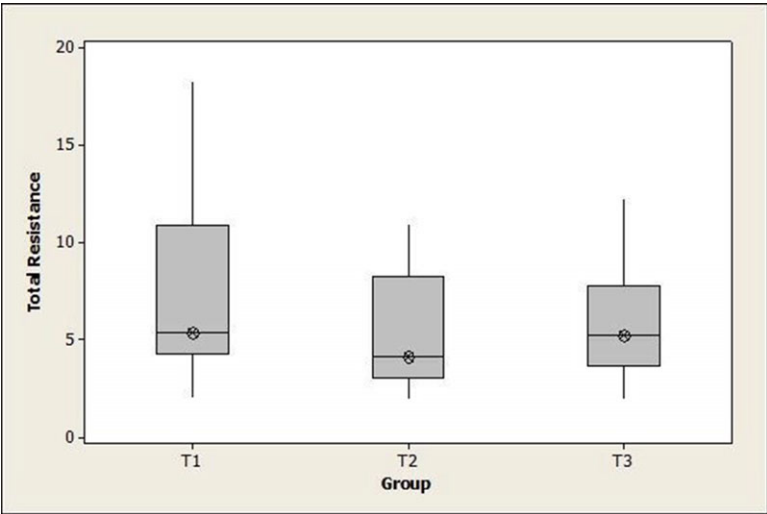


FIGURE 1. Box-blot graphic of total nasal resistances in each trimester.

TABLE 3. The results of ROC curve analysis for total volume, MCA1, and MCA2

	AUC (95% CI)	SE	p
Total volume	0.52 (0.42–0.61)	0.06	0.763
MCA1	0.56 (0.45–0.65)	0.05	0.355
MCA2	0.52 (0.43–0.62)	0.06	0.667

AUC = area under the ROC curve; CI = confidence interval; MCA = minimal cross sectional area; ROC = receiver operating characteristic; SE = standard error.

resistance at mid-cycle demonstrated the effect of raised estrogen levels on the nasal mucosa.²⁷ Estrogen exerts its effects either by increasing the expression of H1 receptors or by changing neurotransmitter concentrations.^{29,30} In contrast, Robinson et al.²⁵ found that increasing levels of beta-estradiol does not cause significant changes in nasal function in women undergoing in vitro fertilization treatment. The authors mentioned that this might be related to the relatively low increase in estrogen compared to pregnancy or due to the noncyclical pattern of estrogen release in contrast to the menstrual cycle.²⁵ The effect of sex hormones on nasal physiology was also studied in postmenopausal women by Caruso et al.²⁶ They conducted a study including 47 postmenopausal women who underwent exogenous hormone therapy. The authors reported that these women had significantly better olfactory sensitivity following hormone therapy. In addition, there was a significant increase in air flow volume and lower nasal resistance during sustained hormone administration.

There is a very limited number of studies in which acoustic rhinometry and anterior rhinomanometry were used to evaluate volumetric changes in pregnant women. Philpott et al.⁷ assessed pathophysiological changes in 18 pregnant women during pregnancy and at 6 weeks postpartum using anterior rhinoscopy, peak inspiratory nasal flow, acoustic rhinometry, anterior rhinomanometry, mucociliary clearance time, and a rhinitis questionnaire. They reported that intranasal combined volume did not decline significantly

during pregnancy. The authors also showed that nasal resistance decreased as pregnancy progressed. In another study, Ellegård et al.³¹ used acoustic rhinometry to assess the superiority of nasal steroids over placebo in 53 pregnant women who had nasal complaints. They did not find significant differences between groups with regard to MCA and volume, but did not report numerical values. Derkay³² reported that pregnant women with symptoms of Eustachian dysfunction (n = 20) had significantly different nasal conductance and inspiratory work/liter as measured by anterior rhinomanometry, compared to a nonpregnant control group (n = 20). In our study, despite the gradual increase in plasma estrogen and progesterone levels throughout pregnancy, the expected influence of these hormones on total nasal volume and nasal resistance was not observed.

The main nasal complaints of pregnant women are stuffiness and congestion similar to other types of rhinitis.^{3,28,33} Pregnant women with nasal congestion have a tendency to mouth breathing at night and their sleep patterns are severely disturbed.^{34,35} As a consequence of mouth breathing, they become regular snorers. In severe cases with OSA, the patients may develop gestational hypertension, preeclampsia and intrauterine growth retardation.^{10,11,18} Sleep breathing disorders in pregnant women may also adversely affect quality of life, especially in the third trimester.^{7,8} In a study by Philpott et al.⁷ the mean score on a rhinitis quality of life questionnaire was worst in the third trimester compared to early gestation and improved

significantly after delivery. Although in that study the symptom score worsened throughout pregnancy, it was not consistent with rhinomanometric findings. However, in our study the NOSE score did not differ between trimesters. This result is more compatible with changes in nasal resistance during pregnancy. Gilbey et al.⁸ conducted a cross-sectional study with 76 pregnant women to assess rhinosinusitis-related quality of life via the 22-item Sino-Nasal Outcome Test (SNOT-22). They reported that the mean item score was significantly higher for the third trimester compared to the second trimester. There was a significant difference between the SNOT-22 scores of third-trimester women and non-rhinosinusitis patients. We found that pregnant women had a lower median NOSE score than the control group. However, only 11.5% of nonpregnant women had a symptom score more than 20, in contrast to 31.7% of pregnant women. This may reflect a negative effect of pregnancy on perception of nasal patency in some women but not in others.

The small sample size and low power of our study are shortcomings. More reliable information could be obtained in a longitudinal study with repeated testing. However, conducting a study with pregnant women presents certain obstacles such as noncompliance, long follow-up duration, and difficulty in retaining pregnant participants. In addition, selecting a control group from pregnant women without nasal complaints rather than nonpregnant women might provide a better chance of correct clinical analysis. Unfortunately, the limited number

of pregnant women without symptoms who accepted to enroll made this unfeasible.

The underlying mechanism explaining why some pregnant women are more vulnerable to severe rhinitis symptoms is not well-known. Differences in circulating hormone levels, mucosal hormone receptor expressivity and sensitivity, and unknown atopy or smoking habits may all contribute to nasal congestion symptoms in pregnant women. We believe that more reliable clinical findings could be achieved in a longitudinal study with a larger sample size. This will allow researchers to exclude individual differences like nasal anatomy, pathophysiology, and subjective symptomatology, which may influence the results.

Conclusion

There is no consensus on the definition, pathophysiology, classification, symptomatology, and treatment of pregnancy-related rhinitis. Whatever the underlying mechanism attributed to it, we found that pregnancy non-significantly decreases nasal cavity volume. However, the symptom score did not change in concert with this reduction. Based on the findings of this study, we concluded that, although there are objective changes in nasal parameters during pregnancy that one can describe as "rhinitis," this clinical entity may not be considered as a disease without complementary symptoms. We believe that further comprehensive studies with larger sample size are required to elucidate the association between pregnancy and the various consequences of nasal congestion. ③

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