

Clinical and Pathogenetic Characteristics of Pregnancy Rhinitis

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Abstract

Pregnancy rhinitis is a very common condition. Defined as "nasal congestion present during 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 wk after delivery," it strikes one in five pregnant women, and it starts in almost any gestational week. The pathogenesis is not clear, but placental growth hormone is suggested to be involved. Smoking and sensitization to house dust mites are probable risk factors. It is often difficult to make a differential diagnosis from sinusitis, which may in pregnancy present with nasal congestion as the only symptom. Antral irrigation is diagnostic for purulent sinusitis and often needs to be repeated, as it should be treated intensively. Because of changes in pharmacokinetics, increased dosage of betalactam antibiotics is needed during pregnancy. As pregnancy rhinitis reduces quality of life and possibly also affects the fetus, there is often need for treatment. Nasal corticosteroids have not been shown to be effective. Systemic administration should be avoided, but nasal corticosteroids could be used in pregnancy when indicated for other sorts of rhinitis. Nasal decongestants give good temporary relief, so pregnancy rhinitis tends to overuse them, giving an additional rhinitis medicamentosa. Therefore, use of nasal decongestants should be restricted to a few days. Invasive methods of turbinate reduction may be effective but are not recommendable in this self-limiting condition because of side effects. Nasal saline washings, exercise, and mechanical alar dilators are safe general means to relieve nasal congestion, but the ultimate treatment remains to be found.

Index Entries

Pregnancy; nasal congestion; rhinitis; treatment; pathogenesis; sinusitis.

Introduction

Pregnancy rhinitis comprises nasal congestion resulting from pregnancy—observed as a phenomenon for ages but until recently not recognized as a defined condition worth being taken seriously.

Air passing in through the nose is prepared to suit the lungs; it is filtered from particles, tempered, and humidified. Nasally inhaled air also transports nitric oxide from the sinonasal unit to the lungs, giving vasodilatation (1). Nasal congestion may force the subject into mouth breathing, which lacks these functions.

It is well known that nasal congestion is increased when the subject is in the supine position, especially in patients suffering from rhinitis (2). A recent paper reports that snoring during pregnancy is very common and that it may have negative effects on the woman as well as on her fetus, such as maternal hypertension, preeclampsia, intrauterine growth retardation, and lower Apgar scores (3). Thus, as nasal congestion may result in snoring, pregnancy rhinitis may cause more serious complications than had earlier been realized.

In the past few decades, the awareness of rhinitis medicamentosa has increased. Women with pregnancy rhinitis tend to use nasal decongestants for prolonged periods of time and are at risk to develop this additional condition, which is characterized by rebound swelling, hyperreactivity, and tolerance (4).

Questionnaires on quality of life have revealed new aspects of disease. Quality of life scores have proved to be worse in allergic rhinitis than in asthma, possibly influenced by problems like day tiredness, thirst, poor concentration, and headache (5). This has not yet been evaluated in pregnancy rhinitis, but a parallel seems obvious.

Nasal congestion increases mouth breathing, which leads to a dry mouth; with less saliva, the dental protection system against caries is disturbed. Longstanding nasal congestion can also induce sinusitis.

Knowledge of effects of nasal congestion is probably confined to ear, nose, and throat (ENT)-oriented physicians, and pregnant women are mainly in contact with obstetricians, who may not be aware of the problem. A patient with a condition like pregnancy rhinitis, concerning two specialties, is at risk to fall between them. Research and information may help to close this gap.

Historical Background

In the late 19th century, a discussion arose about a connection between the genital organs and the nose. In a case report from 1881, Bresgen (6) described a woman with ozaena, which worsened during menstruation. In 1884, Mac Kenzie quoted the Ayurvêda, Hippocrates, Celsus, and several 19th-century writers and presented multiple observations of his own, including “the erection of the nasal turbinated structures” during menstruation, epistaxis as a vicarious menstruation, and worsening of nasal symptoms during menstruation or sexual excitement (7). He expanded his theories and included nasal congestion during pregnancy “at periods corresponding to those of the menstrual flow” (8). Endriss (9) also described epistaxis and worsening of nasal disease resulting from menstruation.

After that rather intense period of publications, the interest seemed to fade until 1943, when Mohun (10) presented 20 cases of “vasomotor rhinitis” during pregnancy appearing in the third to seventh month of gestation, persisting to term, with all but 1 normalizing within 10 d postpartum. He concluded that there was a connection with estrogen but also that “The acromegaly of pregnancy may in some way predispose the nasal structure to vasomotor rhinitis.”

Case reports of success with nasal application of estrogen in the treatment of atrophic rhinitis (11,12) suggested an important influence of estrogen on the nasal mucosa that is possibly also operative in pregnancy rhinitis.

In a summary in 1950, Holmes et al. (13), referring to the predecessors mentioned earlier, stated that "the occurrence of hyperemia with swelling and hypersecretion in the nose as an accompaniment of menstruation does not require further confirmation," and they emphasized the influence of psychological factors on the pathogenesis of those symptoms in menstruation as well as in pregnancy.

Diagnosis

Later studies on the nasal congestion of pregnant women have continued to investigate the phenomenon without stating a proper definition. The common presumption has been that it is a condition that appears at the end of the first trimester and disappears after delivery (14,15). In a study including a control group of nonpregnant women, unspecified subjective nasal congestion was significantly increased only during the third trimester (16).

To define pregnancy rhinitis, we followed 23 women who throughout pregnancy, until 1 mo after delivery, reported their degree of daily nasal congestion and registered nasal peak expiratory flow (nPEF) (17). The women were more congested during than after pregnancy, excluding days with other signs of respiratory tract infection (Fig. 1). In cases where, by our clinical experience, we were convinced that there was a congestion caused by pregnancy, we made the diagnosis. We then looked at the cases and stated the criteria, which would separate those who were diagnosed with pregnancy rhinitis from the rest of the women. This resulted in our clinical definition: "nasal congestion present during 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 wk after delivery." In addition to this, patients often suffer from watery or viscous clear secretion. The swollen mucosa of the nasal turbinates responds to local decongestion, which makes further inspection of the nasal cavities possible (Fig. 2).

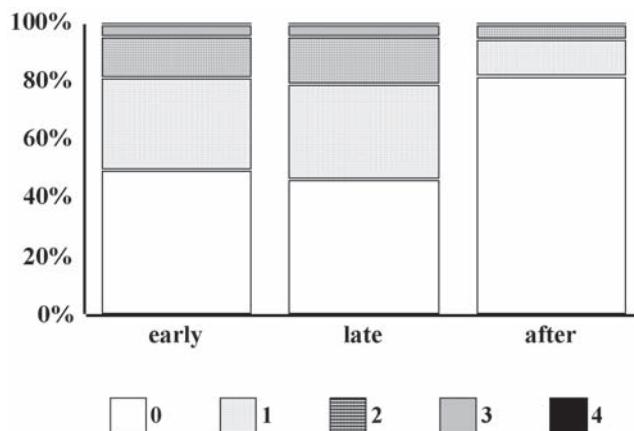


Fig. 1. Subjective nasal congestion scores 0–4 of 23 women during two 4-wk periods; gestational weeks 15–18 (*early*) and the last month preceding delivery (*late*) were significantly higher than the month after delivery (*after*), ($p = 0.001$, Wilcoxon signed rank test).

In our study, the group of women with pregnancy rhinitis had no change in nasal mucociliary transport speed; however, transport speed was found to be significantly decreased during pregnancy in the group of women without the condition (18). A similar physiological reduction was reported in the one other published paper on the subject (19), but further studies on larger populations are needed to confirm the findings and to understand the mechanisms responsible, including the biologic functions of the changes. Measurement of mucociliary transport speed is of no diagnostic value.

Objective measurements of nasal congestion can also be used in studies but are not needed for the clinical diagnosis. Our diagnostic criteria for pregnancy rhinitis include the natural course after delivery to make studies on etiology and epidemiology possible. However, in clinical practice the diagnosis is made once a pregnant woman with nasal congestion not resulting from any other condition makes a consultation for it.

Differential Diagnosis

It is important to exclude sinusitis because it may, in pregnancy, present nasal congestion

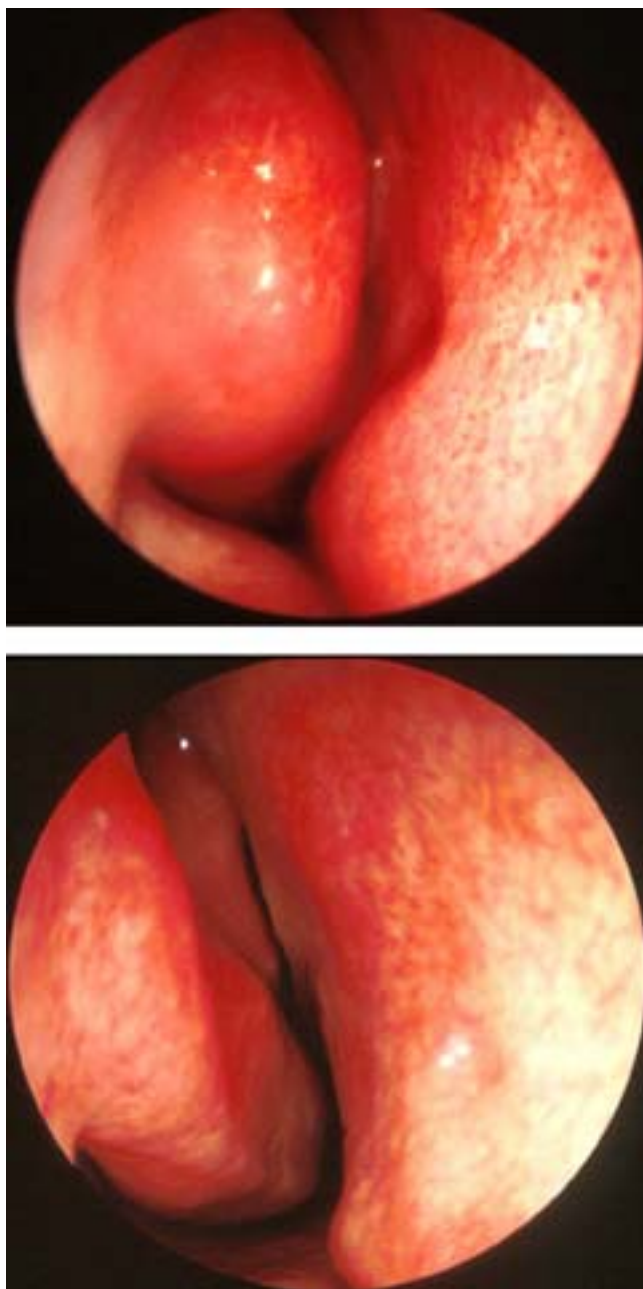


Fig. 2. Endoscopic appearance of the right inferior turbinate in pregnancy rhinitis before and after decongestion.

as the only symptom (20). Even in nonpregnant women, sinusitis is often a difficult diagnosis to make, and the presence of nasal congestion is not of any help in that respect. However, purulent secretion in the middle meatus, sensation of foul smell, unilateral predominance of purulent discharge, and unilateral predomi-

nance of local pain are strong diagnostic signs for sinusitis (21,22). Decongestion facilitates nose inspection, which is best performed with an endoscope, but anterior rhinoscopy is also useful. Ultrasound examination of the sinuses could be an additional tool in experienced hands. In exceptional cases, X-ray may be needed, taking care not to expose the fetus. Antral puncture is the ultimate diagnostic method for sinus empyema.

Nasal congestion resulting from airborne allergy is often combined with watery secretion and sneezing, which is not the case in pregnancy rhinitis. Rhinitis from house dust mites (HDMs) often produces nasal congestion only, and if it appears during pregnancy for the first time, it is difficult to differentiate from pregnancy rhinitis because they may coexist. Allergy is easily excluded by relevant tests for specific IgE.

Rhinitis medicamentosa is a differential diagnosis as well as a complication to pregnancy rhinitis. Because most patients do not spontaneously mention their longstanding use of nasal decongestants, it is important to stress that question. If the nasal congestion persists when the use of decongestants has ceased, then it is no longer caused by rhinitis medicamentosa. In healthy persons with induced rhinitis medicamentosa, the congestion disappears within 2 d after the use of decongestants has stopped (23). However, to be sure in pregnancy rhinitis, 1 wk seems more appropriate.

Nasal granuloma gravidarum (i.e., pregnancy tumor, pregnancy granuloma, telangiectatic polyp) is a benign, rapidly growing tumor that causes nasal obstruction. The histology is practically the same as in pyogenic granuloma. In contrast to pregnancy rhinitis, it is almost always unilateral, and it tends to cause recurrent nosebleed. Inspection of the nasal cavity reveals a lesion that is well vascularized and bleeds easily at the touch. It may even protrude and occupy the nasal vestibulum to be readily seen from the outside. Excision under local anesthesia is indicated if nasal obstruction or

nosebleeds are troublesome, but there is a likelihood of spontaneous regression after delivery (24).

Etiology

Nasal mucosal swelling can be caused by a decrease in the α -adrenergic tonus to the venous sinusoids, giving increased vascular pooling of blood. Another mechanism is through leakage of plasma from the vascular bed into the stroma of the mucosa, giving edema. Neither of these pathways is known to be activated by hormones in the nasal mucosa.

Estrogen

The prevailing theory has been that raised levels of estrogen cause nasal congestion during pregnancy. This is based mainly on the results of Toppozada et al. from biopsy studies on nasal mucosa during pregnancy (15) and from women taking contraceptive pills (25), although a similar study during the menstrual cycle failed to show any cyclic changes (26). The early high-estrogen contraceptive pills produced nasal congestion as a side effect.

The theory of estrogen giving rise to nasal obstruction would imply a relative congestion during the preovulatory and luteal phases of the menstrual cycle, when the serum levels of estrogen are highest. However, in a study of 41 normally menstruating women who registered subjective nasal congestion and nPEF for 3–6 mo, we found significantly more congestion during the menstrual phase, when the estrogen levels are lowest (27). Furthermore, serum levels of estradiol measured four times during pregnancy in 23 women were not shown to be more elevated in women with pregnancy rhinitis than in women without (28).

If nasal congestion were induced by estrogen, rising congestion would be expected in all pregnant women. But only 8 out of 23 pregnant women had increasing congestion, and surprisingly, 9 individuals, registered declining congestion in the course of pregnancy; thus, the further advanced the pregnancy was, the

easier it was to breathe through the nose (17). A parallel is obvious: about one-third of asthmatics improve during pregnancy, whereas one-third deteriorate (29). Although reports of the exact proportions still vary greatly (0–69% and 0–44%, respectively) and are influenced by the different ways asthma severity was assessed and the variable severity of disease in the groups that were studied, the existence of this variation in pregnant asthmatics seems indisputable. The largest cohort studied (568 patients) confirmed these figures and also showed a significant relationship between self-reported change in asthma course and rhinitis during pregnancy (30). However, this is not surprising, as there is a widely accepted connection between asthma and rhinitis in nonpregnants (i.e., the concept of the “united airways” [31], where upper and lower airway inflammatory events influence each other because they are part of a systemic condition with variable manifestations).

Progesterone

Serum levels of progesterone were also similar in both of our groups (28), not supporting the mechanism proposed by Schatz and Zeiger of increased circulating blood volume, possibly enhanced by a vasodilating effect of progesterone being responsible for pregnancy-induced nasal congestion (32). Although no quantitative confirmation of this vascular pooling exists, they do consider it an apparent cause (33).

Placental Growth Hormone

Human growth hormone (hGH) is secreted in episodic bursts. After the first trimester of pregnancy, this is replaced by a continuous secretion with rising values of a placental growth hormone (PGH) variant (34). PGH has an amino acid sequence similar to that of hGH but for 13 amino acid substitutions (35).

Insulin-like growth factor (IGF)-I is associated with the secretion of hGH (36) and is known to rise during the course of pregnancy,

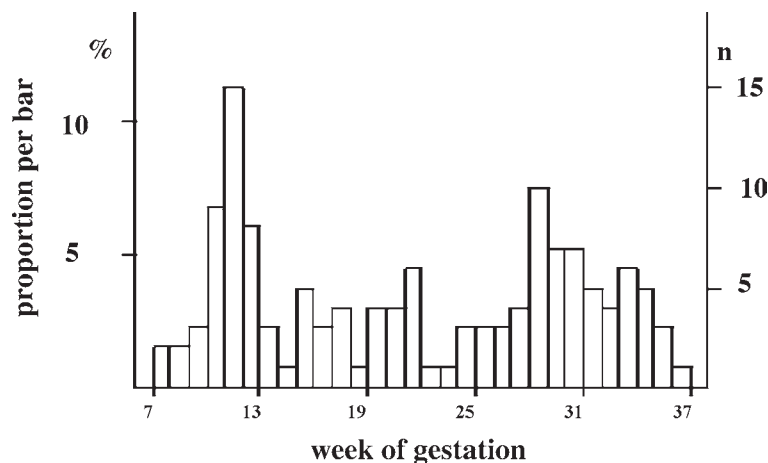


Fig. 3. Pregnancy rhinitis appeared at any time during gestation in the 133 women. For details, see text.

possibly because of increased secretion of PGH (37). IGF-I has been linked to regenerative activity in nasal mucosal cells (38) and to the formation of nasal polyps (39).

In our study, serum levels of PGH were significantly higher in the pregnancy rhinitis group on all occasions throughout pregnancy. Still, the mean values of body weight as well as serum levels of hGH and IGF-I were similar in the two groups (28).

A "hormonal rhinitis" has been suggested to occur in acromegaly (40). The theory that hGH may induce changes in the mucosa of the upper airways is supported by Skinner and Richards (41). They reported a significantly increased frequency of mucosal hypertrophy and polyp formation in the sphenoid and ethmoid sinuses of acromegalic patients compared with patients with prolactinoma. No such pathology was found in the nasal mucosa; however, nasal mucosa was examined after preoperative cocaine treatment. We believe that PGH may stimulate mucosal growth in a similar way and thereby induce pregnancy rhinitis.

Prolactin

The production of prolactin by the pituitary increases during pregnancy and decreases after delivery, suggesting a possible role in the

pathogenesis of pregnancy rhinitis. However, this is contradicted by the absence of sinus pathology in the prolactinoma patients mentioned earlier (41). Furthermore, bromocriptine and quinagolide, which reduce prolactin, both have nasal congestion as known side effects.

Incidence

Earlier reports on incidence comprised small numbers of women; 30% in 79 women (14), 18% in 66 women (42), and 21% in 160 women (43).

We made a questionnaire study on 810 women who registered pregnant during 1 yr in five antenatal centers. The questionnaire concerned nasal congestion upon all visits during and after pregnancy and was completed by 599 women (response rate: 74%) (44). The incidence of pregnancy rhinitis was 22%. It manifested itself in weeks 7 through 36 of gestation (i.e., from as early as it was possible for us to register until as late as possible to obtain a 6-wk duration) (Fig. 3). By excluding women with longstanding nasal problems before conception, we may have underestimated the incidence of pregnancy rhinitis. But as their symptoms may have varied over time in the non-pregnant status, we found it too difficult to evaluate what impact pregnancy may have had.

In another study of 2264 Swedish pregnant women on visits in weeks 12, 20, 30, and 36 of

gestation, a positive answer regarding “daily nasal stuffiness during the last 3 weeks” on either of the visits sorted those women into the “stuffiness” group (45). In week 36, the rate was 42%. Out of 1546 women who answered on all four occasions, 11% reported stuffiness every time. These figures include all types of rhinitis: our figure on pregnancy rhinitis and other sorts of nasal congestion caused, for example, by allergy, overuse of nasal decongestants, or longstanding infections.

Still, there is a recent study that fails to show any significantly increased frequency of nasal congestion during pregnancy (46). The study asked 27 nonpregnant and 33 pregnant women to score nasal congestion on a visual analog scale (VAS) on one occasion in each trimester and compared the “prevalence of congestion.” The cut-off level on the scale that was used to define congestion was not specified in the article. The resulting 33 (nonpregnant), 61, 55, and 55% (each of the trimesters), respectively, was not **significantly** different, in spite of the “congestion” in pregnancy being almost twice as frequent as in the controls. This method, with interindividual comparison of VAS values, is most certainly not appropriate for such a small number of women, but no power calculation was presented in the article.

Risk Factors

Smoking

The incidence of pregnancy rhinitis in our questionnaire study was significantly higher in smokers than in nonsmokers (odds ratio: 1.7; 95% confidence interval 1.1–2.5) (47). On registration, 13% of the women reported themselves as smokers. It is possible that smoking, which is an irritant, adds to other changes in some women and thus produces nasal congestion.

Allergy

The electron-microscopic findings of the nasal mucosa from pregnant women with nasal symptoms were, in the opinion of Toppozada

et al., identical to those in allergic rhinitis (15). Mabry found no connection between anamnestically constant or frequent nasal congestion in pregnancy and previously documented allergic rhinitis (42).

In our questionnaire study, the proposed risk factors asthma, hay fever, and month of conception were not shown to influence the incidence of pregnancy rhinitis (44). In vitro tests for 10 airborne allergens were performed in a study of 165 of those women, 83 of whom had had pregnancy rhinitis (47). The overall sensitization, with or without clinical asthma or rhinitis, was not raised in the group of women who had had pregnancy rhinitis compared with the group of women who had not. However, sensitization to HDMs was more frequent in the former group. Thus, it seems that the few women who have high levels of IgE against HDMs are more likely to get pregnancy rhinitis. It is impossible to separate their pregnancy rhinitis from a subclinical allergic rhinitis with deterioration during pregnancy, but they all clearly recovered after delivery, indicating a pregnancy-related reaction.

Serum levels of soluble intercellular adhesion molecule-1 (sICAM-1) have been shown to be elevated in perennial (48) as well as in seasonal allergic rhinitis (49) and thus can be used as a marker of allergic nasal disease. In our study of 23 pregnant women, the group of women with pregnancy rhinitis had similar mean serum values of sICAM-1 as the group without the diagnosis, and the values did not change significantly over time (47).

Hyperreactivity

A limit of 0.4 mm of ipsilateral congestion measured with rhinostereometry 5 min after provocation with 2 mg/mL histamine has been proposed to differentiate hyperreactive subjects from controls (50). We compared 12 women who had had pregnancy rhinitis in this respect with 13 women who had not (47). There was no difference between the two groups regarding number of cases exceeding that limit, nor

was there any difference between the groups regarding reactions to increasing concentrations of histamine, as measured by rhinostereometry or acoustic rhinometry.

Possible Pregnancy Complications

Effects of pregnancy rhinitis on the fetus seem to be connected with the mother's sleep. It has long been known that nasal congestion increases when the subject is in the supine position, especially in patients suffering from rhinitis (2). Difficulty breathing through the nose increases the tendency to revert to mouth breathing and snoring. Regular snoring was reported in 9% of 73,231 women and was shown to be associated with hypertension, independent of body mass index (51). In a questionnaire study of 502 women the day after delivery, habitual snoring during the previous week was reported in 23% (3). Snorers had a significantly higher frequency of hypertension, preeclampsia, and intrauterine growth retardation, and the Apgar scores of their babies were lower.

In the maxillary sinuses, a continuous production of nitric oxide results in very high concentrations, which constitutes the main source of nasal nitric oxide and when inhaled reduces pulmonary vascular resistance and increases pulmonary oxygenation (52). There is a possibility that mouth breathing resulting from pregnancy rhinitis may affect pulmonary vascular tone and/or oxygenation and possibly the oxygen supply to the fetus. This could be a mechanism behind the complications to snoring described earlier. Furthermore, there is a risk for pregnancy rhinitis to induce development of sleep apnea in women who are predisposed to that disease but who normally can breathe through the nose.

Treatment

Patient Education

As suggested by Rambur (53), on their first prenatal visit all pregnant women should be informed of pregnancy rhinitis and of the

simple treatment measures discussed later, which mostly reduce but do not remove the symptoms. It is important to emphasize that local decongestants should be avoided. Patients can be comforted by knowing that their nasal congestion is a common, self-limiting experience shared by generations of women, noted in ancient writings, and described since the beginning of modern scientific literature. If women expect nasal congestion to accompany pregnancy and are informed of the treatment options, they presumably handle it better once it appears.

Physiological Adjustments

The nasal valve is the narrowest part of the airway, and in alar insufficiency it may be completely blocked upon inspiration. An internal nasal dilator has been shown to significantly reduce snoring and morning tiredness in men during a 6-mo study (54) and is as effective as a local decongestant in healthy subjects (55). An external nasal dilator improved subjective nasal breathing in a study of 24 patients with "pregnancy-related nocturnal nasal congestion," although criteria for the congestion were not specified (43). As there are no side effects to this treatment, it is well worth trying, especially when the nasal congestion disturbs sleep.

Patients may need to be reminded of the simple measure to raise the head end of the bed sufficiently. In the supine position, nasal resistance increased in subjects with rhinitis, but this was not the case in positions where the upper part of the body was elevated above 30° (2) or 45° (56).

Light to moderate exercise has a well-known decongesting effect on the nasal mucosa (57) and may have an additional effect on sleep disturbance because of nasal congestion; the normal fatigue and well-being following exercise may help the woman sleep.

Saline Nasal Washings

Nasal washings with saline solution (5 mL of salt in 0.5 L of water) are, in my clinical experience, helpful for many women with

pregnancy rhinitis as well as in other sorts of rhinitis. The patient is instructed to prepare the washing at home and to lean forward when ingesting it through the nose. She can choose to just sniff it from a cupped hand or to use any of the various products available to make the administration more comfortable. The saline washing produces temporary relief, reduces the amount of secretions, and removes crusts that impair the nasal airway. There are no restrictions as to how often washings may be performed.

Nasal Decongestants

Women with pregnancy rhinitis tend to overuse nasal decongestants because they provide good temporary relief. This results in an even more troublesome situation resulting from rhinitis medicamentosa with rebound swelling, hyperreactivity, and tolerance, which does not resolve after delivery. The patient grows accustomed to the decongested state and has to be informed about the situation (4). Even a dosage given only in the evenings to healthy subjects results in rhinitis medicamentosa (58). For this reason, nasal decongestants should be used in short courses only. Some authors recommend that during pregnancy, nasal decongestants should not be used for more than 5 (33) or even 3 (59) d.

Oral Decongestants

Orally administered vasoconstrictors, such as pseudoephedrine and phenylpropanolamine, are widely used to relieve nasal congestion in various sorts of rhinitis (60). No data are available on whether they are efficient or not in the treatment of pregnancy rhinitis. Recommendations for use during pregnancy when indicated for other diagnoses vary between countries; for example, phenylpropanolamine is the decongestant classified as safe for use during pregnancy in Sweden, and pseudoephedrine is the decongestant preferred in US guidelines (60). Earlier reports on increased risk for the malformation gastroschisis could

not be confirmed in a larger study of 206 cases compared with 798 controls (61). However, as in nonpregnant women, there is a risk for systemic adverse effects including elevated blood pressure, palpitations, loss of appetite, tremor, and sleep disturbance.

Corticosteroids

Corticosteroids have been used for various nasal conditions, but prolonged or repeated use of systemic steroids should generally be avoided to prevent adrenal suppression or other systemic side effects. The effect of nasal steroids in treating allergic rhinitis is indisputable (62,63). They have dramatically reduced the need for systemic steroid treatment. Their efficacy is also documented for treatment of nasal polyps (64), rhinitis medicamentosa (65), and perennial, nonallergic rhinitis (66). However, pregnant patients were never included in any of these studies.

The only earlier published article that deals with steroid treatment for rhinitis resulting from pregnancy is Mabry's (14) description of his experiences with the "gradual but sustained congestion of the nasal mucosa, usually developing toward the end of the first trimester of pregnancy" and its treatment. In his opinion, short courses (≤ 2 wk) of oral corticosteroids may give temporary relief, allowing withdrawal of topical vasoconstrictors, and intranasal submucosal injections of corticosteroids show effects within a few hours and last for 4–6 wk. There are no studies made on these treatments, and the risk for side effects affecting the fetus and the pregnant woman is obvious, including blindness after intranasal injections.

We evaluated the effect of an 8-wk treatment with fluticasone propionate aqueous nasal spray in 53 women with pregnancy rhinitis in a placebo-controlled, randomized, double-blind study with parallel groups (67). Daily symptom scores and nPEF, as well as acoustic rhinometry before and after treatment, did not show any difference between the groups. Placebo resulted in 6 of 27 responders, compared

with 5 of 26 for active treatment. There also was no detectable influence on maternal cortisol as measured by morning S-cortisol and overnight 12-h-U-cortisol or any difference in ultrasound measures of fetal growth or pregnancy outcome.

Using the Swedish Medical Birth Registry, congenital malformations were studied in 2014 infants whose mothers had used inhaled budesonide for asthma during early pregnancy (68). No increase in the rate of congenital malformations was observed compared with the general population rate. Thus, when inhaled nasally, it could probably be used safely in this respect for rhinitis during pregnancy.

Surgery

The use of invasive methods such as electrocautery, cryotherapy, laser, or radiofrequency to reduce mucosal swelling of the inferior turbinates in pregnancy rhinitis is discussed by Rambur (53). Each method has its advantages, as have different types of surgical reduction. The effect may be temporary or permanent, and the frequency, degree, and longevity of side effects such as crusting, edema, and bleeding vary (69). The tendency of pregnancy rhinitis to heal spontaneously after a limited period of time has to be kept in mind. These methods should be carefully considered in desperate cases only, for example, if other sorts of treatment of the nose fail and continuous positive airway pressure is not tolerated in a woman with pregnancy rhinitis and sleep apnea.

Antibiotics

Antibiotics are not indicated in pregnancy rhinitis, but if a sinusitis is found, then it is important to treat it intensively. Repeated antral irrigations are often needed when the sinusitis has been longstanding before diagnosis. The dosage of betalactam antibiotics needs to be elevated by 50% because of increased renal clearance during pregnancy (70). To increase the time over "Minimum Inhibitory Concentration," an extra dose per day is recommended.

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