

Pregnancy Rhinitis

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Pregnancy rhinitis has evolved only lately as a defined condition to be taken seriously, even though nasal congestion that is due to pregnancy has been known as a phenomenon for years. It may seem to be a harmless condition compared with, for example, preeclampsia, which is considered “the commonest cause of maternal and fetal morbidity and mortality,” and occurs in 7% to 9% of the pregnant population [1]. There is a possible connection between pregnancy rhinitis and preeclampsia, which makes the subject even more important to study.

This possible connection is snoring. Snoring is common during pregnancy, and may have negative effects, such as maternal hypertension, preeclampsia, intrauterine growth retardation, and lower Apgar scores [2]. It is well known that nasal congestion increases when the subject is in the supine position, especially in patients who suffer from rhinitis [3], and that nasal congestion may result in snoring.

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 (NO) from the sinonasal unit to the lungs and gives vasodilatation [4]. Nasal congestion may force the subject into mouth breathing, which lacks these functions. Mouth breathing also gives a dry mouth, and with less saliva, the dental protection system against caries is disturbed. Long-standing nasal congestion also can predispose to sinusitis.

Quality of life scores are worse in allergic rhinitis than in asthma, and possibly are influenced by problems like daytime tiredness, thirst, poor concentration, and headache [5]. A parallel to the symptoms of pregnancy rhinitis seems obvious, but this has not been evaluated.

Rhinitis medicamentosa has become recognized as a problem during the last decades. Women who have pregnancy rhinitis tend to overuse nasal deconges-

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tants, and to develop an additional rhinitis medicamentosa, which does not resolve spontaneously after delivery [6].

Knowledge of nasal congestion, such as outlined above, probably is confined to ear, nose and throat- and allergy-oriented physicians, whereas pregnant women are mainly in contact with their obstetricians, who may not be aware of the problem.. Research and education should increase the knowledge of this problem by health care professionals and patients, and lead to improved management and quality of life. .

Historical knowledge

In the late nineteenth century, several scientific papers reported observations that connected the female genitals with the nose. A case report from 1881 [7] described a woman who had ozena, which worsened during menstruation. In 1884, Mac Kenzie [8] quoted the Ayurvêda, Hippocrates and Celsus, and several nineteenth century writers, and presented multiple observations of his own on “the erection of the nasal turbinated structures” during menstruation, and worsening of nasal symptoms during menstruation or sexual excitement. Expanding his theories, he included nasal congestion during pregnancy in 1898 [9]. In 1892, Endriss [10] described epistaxis and worsening of nasal disease that was due to menstruation.

After this period, the interest seems to have faded until 1943, when Mohun [11] presented 20 cases of “vasomotor rhinitis” during pregnancy. Nasal symptoms in these patients appeared in the third to seventh month of gestation, persisted to term, and normalized in all but one patient within 10 days post partum. 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.”

Estrogen was assumed to influence the nasal mucosa, and there were case reports of successful nasal estrogen treatment of atrophic rhinitis [12,13]. Holmes and colleagues [14] stated in 1950, that “the occurrence of hyperemia with swelling and hypersecretion in the nose as an accompaniment of menstruation does not require further confirmation.” They emphasized the influence of psychologic factors on the pathogenesis of those symptoms in menstruation as well as in pregnancy.

Possible influence of rhinitis on the fetus

It is well known that nasal congestion increases when the subject is in the supine position, especially in patients who suffer from rhinitis [3]. Difficulty breathing through the nose increases the tendency to revert to mouth breathing, and snoring. Regular snoring, reported in 9% out of 73,231 nonpregnant women, was associated with hypertension independent of body mass index [15]. In a questionnaire study in 502 women the day after delivery, habitual snoring the

previous week was reported in 23% [2]. Snorers had a significantly higher frequency of hypertension, preeclampsia, and intrauterine growth retardation, and the Apgar scores of their babies were lower.

Inhaled NO is produced mainly in the maxillary sinuses. It reduces pulmonary vascular resistance and increases pulmonary oxygenation [16]. Mouth breathing that is due to pregnancy rhinitis may reduce this inhalation, affect pulmonary vascular tone or oxygenation, and thus, affect the oxygen supply to the fetus. This could be a mechanism of the complications that are associated with snoring that were described above.

Furthermore, there is a risk that pregnancy rhinitis induces obstructive sleep apnea in women who are predisposed to that disease, but who normally can breathe through the nose. Hypertension that is associated with preeclampsia is characterized by an increase in nocturnal blood pressure, a pattern that was reported to be associated with snoring and obstructive sleep apnea [1].

Definition for research purposes

Many investigators have studied the nasal congestion of pregnant women without a proper definition of the phenomenon. Some have described it as a condition that appears at the end of the first trimester and disappears after delivery [17,18]. Another study found that unspecified subjective nasal congestion was increased significantly only during the third trimester compared with nonpregnant subjects [19].

With the aim to define pregnancy rhinitis, we followed 23 women, who scored nasal congestion and registered nasal peak expiratory flow (nPEF) daily until 1 month after delivery [20]. The women were more congested during pregnancy, than after pregnancy, excluding days with other signs of respiratory tract infection

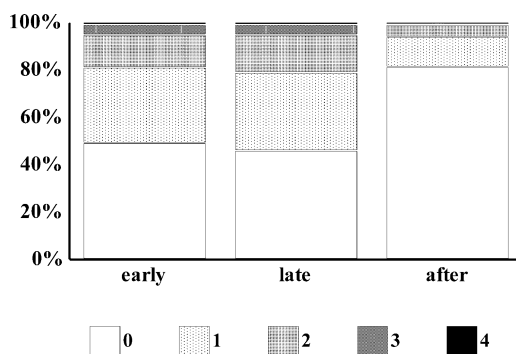


Fig. 1. Subjective nasal congestion scores (0–4) of 23 women during three 4-week periods. Scores in gestational weeks 15–18 (early), and in the last month preceding delivery (late) were significantly higher than in the month after delivery (after) ($P = .001$, Wilcoxon signed rank test). (From Ellegård E. Clinical and pathogenetic characteristics of pregnancy rhinitis. Clin Rev Allergy Immunol 2004; 26:149–59; with permission.)

(Fig. 1). In cases where, by our clinical experience, we were convinced that there was nasal congestion caused by pregnancy, we made the diagnosis. We then looked at the cases, and identified the criteria that would separate them 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 weeks after delivery.” These diagnostic criteria for pregnancy rhinitis include the natural course after delivery, to make studies on etiology and epidemiology possible.

Nasal mucociliary transport speed was not affected in the group of women who had pregnancy rhinitis; however, it was decreased significantly during pregnancy in the group of women who did not have the condition [21]. A similar [22], and a reverse [23] physiologic change have been reported, which suggests that further research is needed.

Diagnosis

The clinical diagnosis of pregnancy rhinitis can be made once a pregnant woman who has nasal congestion that is not due to any other condition presents for care. In addition to congestion, patients often suffer from watery or viscous clear secretion from the nose. The swollen mucosa of the nasal turbinates responds to local decongestion, which makes complete inspection of the nasal cavities possible (Fig. 2). Objective measurements of nasal congestion can be used in scientific studies, but are not needed for the clinical diagnosis. Measurement of mucociliary transport speed is of no diagnostic value.

Differential diagnosis

Rhinitis medicamentosa is a differential diagnosis (Box 1) as well as a complication to pregnancy rhinitis, because nasal decongestants give good temporary relief. Most patients do not mention their long-standing use of nasal decongestants spontaneously, and it is important to question them about it. In nonpregnant persons who have rhinitis medicamentosa, the rebound congestion usually disappears within 2 days after the use of nasal decongestants has stopped [24]. In pregnancy, clinical experience suggests it more commonly takes 1 week to resolve.

Sinusitis (see the article elsewhere in this issue) may be a differential diagnosis and a complication of pregnancy rhinitis. Even in nonpregnant patients, sinusitis often is a difficult diagnosis to make, and the presence of nasal congestion lacks specificity in that respect. Pus in the middle meatus, sensation of foul smell, unilateral predominance of purulent secretion, and unilateral predominance of local pain have been suggested as strong diagnostic signs for sinusitis [25], but these signs are more specific than sensitive [26]. Furthermore, in pregnancy, nasal congestion may be the only symptom of sinusitis [27]. Decongestion facilitates

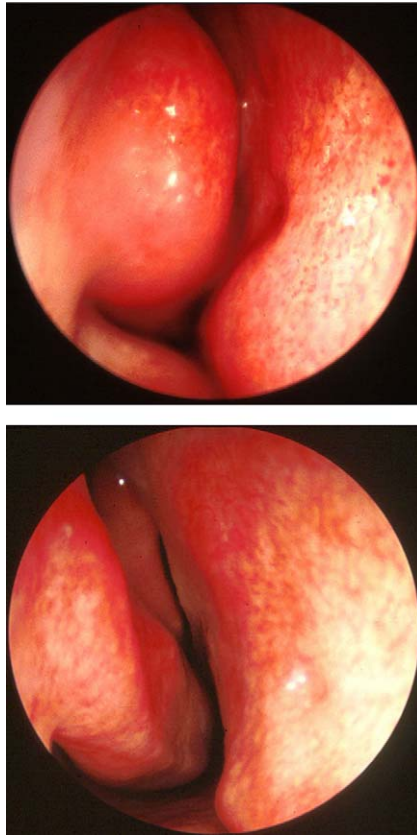


Fig. 2. Endoscopic appearance of the right inferior turbinate in pregnancy rhinitis before (*top*) and after (*bottom*) decongestion. (From: Ellegård E. Clinical and pathogenetic characteristics of pregnancy rhinitis. Clin Rev Allergy Immunol 2004;26:149–59; with permission.)

nose inspection, which is best performed with an endoscope, but anterior rhinoscopy also is useful. Ultrasound examination of the sinuses can be an additional tool in experienced hands. In exceptional cases, a radiograph may be needed, taking care not to expose the fetus. Antral puncture is the ultimate diagnostic method for sinus empyema.

Box 1. Differential diagnosis

- Rhinitis medicamentosa
- Sinusitis
- Upper respiratory tract infection
- Airborne allergy
- Nasal granuloma gravidarum

Upper respiratory tract infection other than sinusitis is more obviously infective, is not confined to nasal congestion, and is not as long-standing.

Airborne allergy is another common differential diagnosis. Nasal congestion in patients who have allergic rhinitis is accompanied most often by excessive watery secretion, and sneezing, which is not the case in pregnancy rhinitis. Allergic rhinitis that is due to house dust mites, however, frequently presents with nasal congestion as the most prominent symptom. If it appears during pregnancy for the first time, it is difficult to differentiate from pregnancy rhinitis, and the two conditions may coexist. Relevant in vitro blood tests for specific IgE excludes allergy.

Nasal granuloma gravidarum (also known as pregnancy tumor, pregnancy granuloma, telangiectatic polyp) is a rapidly growing benign tumor that causes nasal obstruction. The histology is nearly the same as in pyogenic granuloma. In contrast to pregnancy rhinitis, it almost always is unilateral, and it tends to cause recurrent nosebleed. Inspection of the nasal cavity reveals a well-vascularized lesion that bleeds easily upon touch. It even may protrude and occupy the nasal vestibulum and be seen readily from the outside. If nasal obstruction or nosebleeds are troublesome, excision under local anesthesia is indicated, but it may disappear completely by itself after delivery [28].

Etiology

Nasal mucosal swelling can be caused by increased vascular pooling of blood that is caused by a decrease in α -adrenergic tone in the venous sinusoids, or by edema that is caused by leakage of plasma from the vascular bed into the stroma. No hormone is known to activate any of these pathways in the nasal mucosa.

Estrogen

The theory that estrogen causes nasal congestion mainly is based scientifically on the results of Toppozada and colleagues [18] from biopsy studies on nasal mucosa in pregnancy, and from women who were taking contraceptive pills [29], although a similar study during the menstrual cycle failed to show any cyclic changes [30]. Nasal congestion was a known side effect of the early high-estrogen contraceptive pills.

If estrogen causes nasal congestion, this should be found during the pre-ovulatory and luteal phases of the menstrual cycle, when the serum levels of estrogen are highest. In a study over several months on 41 normally menstruating women, significantly more congestion was found during the menstrual phase, when the estrogen levels are lowest [31]. Furthermore, serum levels of estradiol that were measured four times during pregnancy in 23 women were not higher in women who had pregnancy rhinitis [32]. Moreover, recent studies on postmenopausal women suggest that estrogen replacement therapy may reduce subjective nasal congestion [37].

If estrogen causes nasal congestion, one would expect increasing congestion in every pregnant woman. In 23 pregnant women, only 8 had increasing congestion, and 9 individuals registered declining congestion in the course of pregnancy [20]. A similar pattern was seen in a recent study, although the mean results of different measurements indicated increasing congestion during pregnancy [23]. A parallel is obvious: about one third of asthmatics improve during pregnancy, whereas one third deteriorate [33]. Although reports of the exact proportions vary, the existence of this variation in pregnant asthmatics seems to be indisputable [34].

A study on 568 patients also showed a significant relationship between the self-reported change in asthma course during pregnancy, and that of rhinitis [35]. This agrees with the well-known concept of the “united airways” in nonpregnant patients, where upper and lower airway inflammatory events influence each other, because they are part of a systemic condition with variable manifestations [36].

Progesterone

Increased circulating blood volume, possibly enhanced by a vasodilating effect of progesterone, was suggested as being responsible for pregnancy-induced nasal congestion [38]. Serum levels of progesterone were similar in groups of women who did and did not have pregnancy rhinitis, which does not support this theory [32].

Prolactin

The pituitary increases production of prolactin during pregnancy, which suggests a possible role in the pathogenesis of pregnancy rhinitis; however, this is contradicted by the absence of sinus pathology in patients who have prolactinoma [39]. Furthermore, bromocriptine and quinagolide, which reduce prolactin, have nasal congestion as known side effects.

Neuropeptides

Vasoactive intestinal polypeptide (VIP) is associated with other sorts of rhinitis [40], and has been proposed as a possible mediator of the vasodilation in the nasal mucosa that is responsible for nasal congestion during pregnancy. A study on serum levels did not support this theory [41].

Nasal biopsies of postmenopausal women showed increased immunopositivity for estradiol, estradiol receptor, VIP, and substance P (SP) after 6 months of hormone replacement therapy. There was a reduction in neuropeptide Y (NPY). Nasal application induced stronger changes than did transdermal application of hormone replacement therapy. Mucociliary transport time and subjective nasal congestion decreased, but anterior rhinomanometry was unchanged. The investigators proposed that estrogen action in the nasal mucosa is mediated by neuropeptides—an increase of gland secretion and vasodilatation by VIP and SP, and a decrease of NPY-induced vasoconstriction [37]. Other than the one study

that was mentioned above [41], neuropeptides have not been evaluated in patients who have pregnancy rhinitis.

Placental growth hormone

After the first trimester of pregnancy, the episodic bursts of human growth hormone (hGH) are replaced by a continuous secretion with increasing values of a placental growth hormone variant (PGH) [42]. In our study, serum levels of PGH were significantly higher in the group that pregnancy rhinitis on all occasions throughout pregnancy [32].

In acromegaly, the possibility of a “hormonal rhinitis” has been suggested [43]. A study by Skinner and Richards [39] supports this theory: that hGH may induce changes in the mucosa of the upper airways. In their study, patients who had acromegaly had an increased frequency of mucosal hypertrophy and polyps in the sinuses compared with patients who had prolactinoma. No such pathology was found in the nasal mucosa, however, which was examined after preoperative cocaine treatment. It is possible that PGH may stimulate mucosal growth in a similar way, and thereby, induce pregnancy rhinitis. If PGH were the single cause of pregnancy rhinitis, it would be expected to occur in all pregnant women. Further studies will be necessary to determine the etiology of pregnancy rhinitis.

Incidence

Earlier incidence figures were based on small numbers of women: 30% in 79 women [17], 18% in 66 women [44], and 21% in 160 women [45]. In a questionnaire study, of 810 women who were asked about subjective nasal congestion upon all visits to the midwife during and after pregnancy, pregnancy rhinitis was present in 22% of the 599 women who completed the study [46]. Nasal congestion manifested itself in gestational weeks 7 through 36 (ie, from as early as it was possible to register, until as late as possible to obtain a 6-week duration; Fig. 3). The incidence of pregnancy rhinitis may have been underestimated, because women who had long-standing nasal problems before conception were excluded. But because their symptoms may have varied over time in the nonpregnant state, it was too difficult to evaluate what impact pregnancy may have had.

In another Swedish study, 2264 pregnant women were asked whether they had had “daily nasal stuffiness during the last 3 weeks” on visits in gestational weeks 12, 20, 30, and 36. Women with any positive answer made up the “stuffiness” group, which then included all sorts of rhinitis. The “stuffiness” rate was 42% in week 36 of gestation. Out of 1546 women who answered on all four occasions, 11% reported “stuffiness” every time [47].

One study failed to show any significantly increased frequency of nasal congestion during pregnancy [48]. The investigators asked 27 nonpregnant women and 33 pregnant women once in each trimester to score nasal congestion

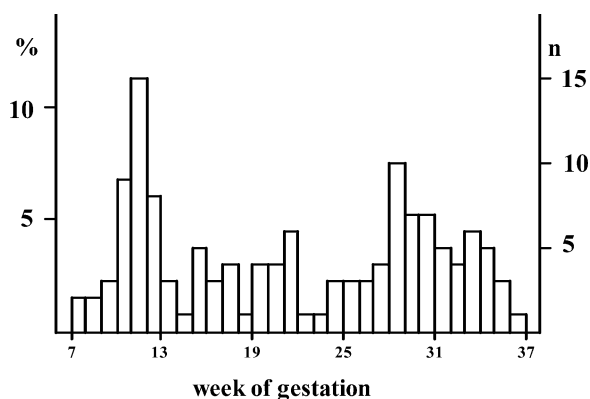


Fig. 3. Debut of nasal congestion due to pregnancy rhinitis in 133 women. (From: Ellegård E, Hellgren M, Torén K, et al. The incidence of pregnancy rhinitis. *Gynecol Obstet Invest* 2000;49: 98–101; with permission.)

on a visual analog scale and compared the “prevalence of congestion.” The cut-off level that was used to define congestion was not specified in the article. The resulting 33% (nonpregnant), 61% (first trimester), 55% (second trimester), and 55% (third trimester) were not reported to be significantly different (despite that “congestion” in pregnancy was almost twice as frequent as in the controls). Their method is unlikely to be appropriate for such a small number of women, but no power calculation was presented.

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) [49]. It is possible that the irritating effects of smoking are additive to other changes that induce nasal congestion.

Allergy

Toppozada and colleagues [18] reported that the electron microscopic findings from nasal mucosa from pregnant women who had nasal symptoms were identical to those in allergic rhinitis. Mabry [44] found no connection between anamnestic constant or frequent nasal congestion in pregnancy, and previously documented allergic rhinitis.

Asthma, hay fever, and month of conception were not associated with pregnancy rhinitis in our questionnaire study [46]. We performed in vitro tests for 10 common airborne allergens on 165 of those women, 83 of whom had had

pregnancy rhinitis [49]. The overall sensitization rate was not increased in the group of women who had had pregnancy rhinitis; however, sensitization to house dust mites was more frequent in that group. Thus, the few women who have high levels of IgE against house dust mites seem predisposed to get pregnancy rhinitis. It was impossible to differentiate their rhinitis from a subclinical allergic rhinitis with deterioration during pregnancy, but it was remarkable that they all recovered after delivery.

Serum levels of soluble intercellular adhesion molecule-1 (sICAM-1) are elevated in perennial [50], and seasonal allergic rhinitis [51], and can be used as a marker of allergic nasal disease. In our study of 23 pregnant women, the mean serum values of sICAM-1 did not change significantly over time, and the group of women who had pregnancy rhinitis had similar values as did the group that did not have the diagnosis [49].

Nasal hyperreactivity

To differentiate subjects who have nasal hyperreactivity from controls using rhinostereometry, a limit of 0.4 mm ipsilateral congestion 5 minutes after provocation with histamine 2 mg/mL has been proposed [52]. We evaluated nasal hyperreactivity that was determined in this way in 12 women who had had pregnancy rhinitis and 13 women who had not had pregnancy rhinitis [49]. There was no difference between the two groups. In addition, there were no differences between the groups regarding reactions to increasing concentrations of histamine, as measured by rhinostereometry or acoustic rhinometry.

Treatment

Antenatal care information

Women who know that pregnancy can induce nasal congestion and are informed of the treatment options presumably handle it better if it appears. Patients are less likely to be worried if they know that their nasal congestion is a common, self-limiting experience, which they share with generations of women. As Rambur [53] suggested, information on pregnancy rhinitis should be given to all pregnant women on their first antenatal care visit (Box 2). This requires an educational effort that is directed toward the antenatal staff as well.

Physiologic measures

Physical exercise has a well-known decongesting effect on the nasal mucosa [54]. The normal fatigue and well being that follow exercise also may have an additional positive effect on sleep disturbance that is caused by nasal congestion.

“Raise the head end of the bed when your nose is congested”—most patients have heard this simple advice but they may need to be reminded of it. The

Box 2. Treatment considerations*General*

Information to all pregnant women
Physical exercise
Raised head end of bed
Mechanical nasal alar dilation
Nasal saline washings

Special indications

Nasal decongestants
Nasal corticosteroids
Nasal continuous positive airway pressure
Invasive inferior turbinate reduction

Not indicated

Systemic corticosteroids
Oral decongestants
Antibiotics

effective angle is 30° [3] or 45° [55]. It is easier to tolerate “books under the legs of the head end of the bed” than extra pillows, when the side position is desired. In pregnancy, that position is favorable because of the vena cava syndrome, and because it reduces the risk of snoring.

Different mechanical devices are available to dilate the nasal valve region, which is the narrowest part of the airway. An external type of device improved subjective nasal breathing in patients who had “pregnancy-related nocturnal nasal congestion” [45]. An internal type of device reduced snoring significantly in men [56], and was as effective as a nasal decongestant in healthy subjects [57]. Because the possible problems that are associated with the use of these devices are limited to local irritation of the skin by glue or pressure, they are well worth trying, especially when nasal congestion disturbs sleep. Nasal valve muscular training [58] might prove to be another effective way to improve nasal breathing.

Nasal saline washings

In the author’s clinical experience, nasal washings with physiologic saline solution (5 mL of salt in 0.5 L of water) are effective in reducing symptoms for many women who have pregnancy rhinitis; they also work well for other sorts of rhinitis. The patient prepares the solution at home, and leans forward when

taking it through her nose. She can sniff it in from her cupped hand, or she can use any of the products that are available to make the administration more comfortable. There are no restrictions on how often it may be used. The saline gives temporary relief, reduces the amount of secretions, and removes crusts that impair the nasal airway.

Nasal decongestants

Nasal decongestants give good temporary relief in pregnancy rhinitis. But because the condition does not resolve in a few days, like a common cold does, pregnant women who have nasal symptoms tend to use them for prolonged periods of time. This results in the additional condition of rhinitis medicamentosa, which does not resolve spontaneously after delivery. Rebound swelling of the mucosa increases nasal congestion when the decongestive effect of the drug has disappeared. To alleviate this symptom, patients gradually use larger doses of the vasoconstrictor more frequently. The preservative, benzalkonium chloride, in the nasal preparation worsens the congestion [59]. Many patients are unaware of the condition. They get used to the decongested state as being normal, and they need to be informed about the situation [6]. Even a dosage that was given only in the evenings to healthy subjects resulted in rhinitis medicamentosa [60]. Some investigators recommend that during pregnancy, nasal decongestants should not be used for more than 5 days [38] or even 3 days [61]. It is possible that lower concentrations and unilateral, alternating nostril administration may be used safely only in the evening for longer periods of time, but this needs to be studied.

Oral decongestants

Orally administered vasoconstrictors, such as phenylpropanolamine and pseudoephedrine, are used widely in various sorts of rhinitis [62]. No data are available on whether they are effective in pregnancy rhinitis. The recommendations for their use during pregnancy vary between countries. For example, phenylpropanolamine is the oral decongestant that is classified to be safe for use during pregnancy in Sweden, whereas pseudoephedrine is preferred in U.S. guidelines [62]. Earlier reports indicated an increased risk for the malformation gastroschisis, but this could not be confirmed in a larger study on 206 cases and 798 controls [63]. It seems unwise to risk the general systemic adverse effects of oral decongestants, which may include elevated blood pressure, palpitations, loss of appetite, tremor, and sleep disturbance.

Nasal corticosteroids

Nasal corticosteroids are effective in allergic rhinitis [64,65]; rhinitis medicamentosa [66]; perennial, nonallergic rhinitis [67]; and nasal polyps [68]. It is important to inform the patient that the anticipated effect is not as immediate and

obvious as that of nasal decongestants. Pregnant patients were not included in any of these studies, and the documentation of treatment during pregnancy is limited.

Fluticasone propionate nasal spray did not show any effect on pregnancy rhinitis in 53 women treated for 8 weeks in a placebo-controlled, randomized, double-blind study with parallel groups, as evaluated by daily symptom scores, nPEF, and acoustic rhinometry [69]. There also were no detectable influences on maternal morning S-cortisol and overnight 12-h-U-cortisol, nor any difference in ultrasound measures of fetal growth, or pregnancy outcome.

Budesonide that was inhaled for asthma by women in early pregnancy did not increase the rate of congenital malformations in 2014 infants compared with the general population rate, according to figures from the Swedish Medical Birth Registry [70]. Thus, it probably also could be used safely in this respect when inhaled nasally for rhinitis during pregnancy. Published data show that the currently available inhaled steroids used at clinically relevant doses do not impair intrauterine growth [71].

Systemic corticosteroids

Corticosteroids have been administered systemically for various nasal conditions, but prolonged or repeated use should be avoided to prevent adrenal suppression, and other systemic side effects. Mabry [17] described his experience in treating congested nasal mucosa in pregnancy. In his opinion, up to 2 weeks' duration of oral corticosteroids may give temporary relief and allow the withdrawal of nasal decongestants, and intranasal submucosal injections of corticosteroids produced improvement within a few hours that lasted for 4 to 6 weeks. No rigorous clinical studies have been published on the safety or efficacy of these treatments during pregnancy, and the side effects from intranasal steroid injections include blindness.

Antibiotics

Pregnancy rhinitis is not an indication for treatment with antibiotics; however, sinusitis during pregnancy should be treated with an elevated dosage of antibiotics (see elsewhere in this issue). Because of increased renal clearance, the dosage of betalactam antibiotics needs to be increased by 50% [72]. An extra dose per day is recommended, to increase the time over "minimum inhibitory concentration." Repeated antral irrigations may be needed when the sinusitis has been long-standing. This is facilitated by the use of a narrow catheter, which is left at the puncture site for a few days (eg, SinoJect).

Nasal continuous positive airway pressure

Nasal continuous positive airway pressure (CPAP) is not indicated in isolated pregnancy rhinitis, but it can be considered in cases of obstructive sleep apnea. As in nonpregnant patients, nasal CPAP has been used with excellent compliance

for that diagnosis, and was shown to be effective as measured by polysomnography. Readjustment of the CPAP pressure may be needed during the course of pregnancy [73].

In a noteworthy study, although not randomized, nasal CPAP reduced nocturnal blood pressure significantly in women who had preeclampsia, although they did not have obstructive sleep apnea [1].

Surgery

Noninvasive methods, such as electrocautery, cryotherapy, laser, or radio-frequency, can be used to reduce mucosal swelling of the inferior turbinates in pregnancy rhinitis [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 persistence of side effects (eg, crusting, edema, bleeding) varies [74]. The spontaneous healing of pregnancy rhinitis after delivery must be kept in mind. These methods should be considered carefully in desperate cases only (eg, if other sorts of treatment of the nose fail and CPAP is not tolerated in a woman who has pregnancy rhinitis and obstructive sleep apnea).

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