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Drug-induced rhinitis as a complication of using mucosal decongestants

Polekowy nieżyt nosa jako powikłanie stosowania leków obkurczających błonę śluzową

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Abstract

Rhinitis medicamentosa is induced by the overuse of topical nasal decongestants. It is characterized by chronic nasal obstruction, not related to allergy or common cold. This problem often affects young and middle-aged patients who, in order to reduce nasal congestion, exudation and swelling of the nasal mucosa, use drugs that only initially relieve the patient's symptoms. Rhinitis medicamentosa is associated with the risk of many complications, including the development of nasal polyps, otitis and sinusitis. The basis of treatment is discontinuation of decongestants and the use of nasal corticosteroid. In situations where conservative treatment is not sufficient, surgical methods can also be used. It is complicated to cure and the therapy is often long-lasting. Therefore, it seems much easier to prevent the development of rhinitis medicamentosa by avoiding this type of drugs or using nasal decongestant only for short periods and in correct doses.

Key words: Rhinitis medicamentosa, Nonallergic rhinitis, Nasal congestion, Decongestants

Introduction

Rhinitis medicamentosa (RM) is a seemingly benign clinical condition, however, it can significantly reduce the quality of life. It is classified as non-allergic rhinitis that occurs due to the use of nasal decongestants.

It is recognized that individuals suffering from chronic nasal obstruction due to conditions such as nasal septum deviation, nasal polyps, vasomotor or allergic rhinitis, are more

susceptible to developing rhinitis medicamentosa. On the other hand, most patients who see a doctor for this condition have developed RM due to improper treatment of colds or other upper respiratory tract infections and prolonged use of decongestants, despite having no prior respiratory problems.

According to reports, this problem most often affects younger patients, mainly those in their third and fourth decades of life, with a comparable incidence in both sexes. It is worth noting the high frequency of respiratory symptoms in children, which undoubtedly places them at risk of developing rhinitis medicamentosa [1].

Medications that can cause rhinitis medicamentosa include imidazole derivatives (e.g. xylometazoline) or sympathomimetic amines (e.g. phenylephrine, ephedrine). These substances help alleviate the symptoms of the illnesses mentioned above by reducing congestion, exudation, and swelling of the mucous membrane.

Despite the undeniable positive effects on the course of many diseases, prolonged use of decongestants can induce the occurrence of rhinitis medicamentosa, also known as the so-called rebound congestion. It is believed that regular use of topical alpha-adrenergic agonists for as little as five days can promote the development of this condition, although this varies individually, with some cases appearing only after several weeks of use.

According to available data, the prevalence of this phenomenon is estimated at 1-2%, with an upward trend noted in recent years [2]. The reasons for this can be attributed to the dynamic development of allergic diseases and, consequently, the increased use of topical nasal decongestants. It is encouraging that the symptoms are usually quickly alleviated after using these medications, allowing individuals to resume their activities shortly thereafter. These medications are available over the counter, making them easily accessible to children [3].

The aim of the work

The aim of the study was to evaluate the relationship between the occurrence of drug-induced rhinitis and the use of mucosal decongestants.

Materials and Methods

The article presents a systematic review of scientific research focusing on the relationship between drug-induced rhinitis and using mucosal decongestants. For this purpose, a database analysis was performed - among others, Web of Science, PubMed, and Google Scholar. Additionally, the bibliography of cited works was reviewed. The review of the literature and article selection were carried out in March 2024.

Pathogenesis

There are several hypotheses explaining the mechanism of rebound congestion, although its exact pathophysiology remains unknown. One suggests that chronic vasoconstriction results in ischemia of the mucous membrane, which in turn promotes its swelling [4]. This was observed during experiments with guinea pigs that were administered naphazoline for four months. Also worth mentioning is a study involving patients suffering from RM, in which congestion of the inferior turbinate was noted on the first day after discontinuation of decongestants. After 14 days of using intranasal steroids instead, the swelling was minimized [5].

Another hypothesis posits the influence of increased parasympathetic activity on the vasomotor neuron. This situation is secondary to reduced endogenous norepinephrine production via a negative feedback mechanism. Chronic use of alpha-adrenergic agonists also reduces the sensitivity of adrenergic receptors, impairing the sympathetic nerves' ability to maintain vascular constriction. As a result of a pathological reaction of the autonomic system, vasodilation and secondary transudation into the submucosal layer occur, leading to its swelling [6].

In one study examining this situation, plasma cells were found to accumulate around the impaired nerve endings in the nasal mucosa of individuals with rhinitis medicamentosa. Histological examination revealed inflammatory infiltrates, increased number of lymphocytes and fibroblasts, hypertrophy of goblet cells, and loss of epithelial cilia [7].

One theory also suggests a delayed and prolonged response of beta receptors to topical decongestants. According to this theory, the dilatory action on beta receptors ultimately lasts longer than the constrictive action on alpha receptors, leading to rebound congestion.

Drugs

The primary cause of rhinitis medicamentosa can be identified as the topical nasal decongestants mentioned above. Two main groups of these medications can be distinguished: sympathomimetic amines and imidazole derivatives.

The former act on alpha-1 and, to a lesser extent, beta receptors. By affecting the sympathetic nervous system through the presynaptic release of norepinephrine, their use results in vasoconstriction. This leads to the desired effect of reduced blood flow, consequently reducing swelling and nasal discharge. Since their affinity for beta receptors is much weaker, when the vasoconstrictive action ceases, reflex vasodilation occurs. For this reason, after short-term relief, the patient feels the need to reapply the medication [8].

Imidazole derivatives, on the other hand, act postsynaptically on alpha-2 adrenergic receptors. Their use leads to decreased production of endogenous norepinephrine through a negative feedback mechanism [9]. With chronic use, the sympathetic nerves may become unable to maintain vasoconstriction due to the ineffectiveness of norepinephrine. Chronic use, such as with oxymetazoline, also reduces the sensitivity of alpha-2 receptors, which manifests as tachyphylaxis—the need for increased doses and/or frequency of application for the medication to continue to be effective [10]. It is believed that after discontinuation of the medication, the vasomotor nerves are unable to maintain proper function, and the deficiencies in norepinephrine result in the predominance of parasympathetic function, leading to rebound congestion. Chronic overuse of decongestants leads to changes in the nasal mucosa, such as destruction of ciliated cells, squamous cell metaplasia, and goblet cell hypertrophy [11].

The term "rhinitis medicamentosa" is also used to describe the phenomenon of nasal mucosal swelling induced by substances other than topical decongestants. Such substances include oral

contraceptives, type 5 phosphodiesterase inhibitors, antipsychotic drugs, and antihypertensive medications [12]. Since a different mechanism underlies the development of this condition, it is proposed that the term "drug-induced rhinitis" be used to describe it.

Symptoms and complications

The most commonly reported symptom by patients is chronic nasal obstruction caused by mucosal swelling [13]. This is often associated with the need to breathe through the mouth, which can lead to dry mouth, increased susceptibility to upper respiratory tract infections, and olfactory disturbances [14]. Typically, there is no nasal discharge, postnasal drip, or sneezing [15]. Non-physiological airflow may also be associated with snoring, sleep problems related to frequent nighttime awakenings, and subsequently, headaches and fatigue [16].

Effective treatment of rhinitis medicamentosa is crucial due to the risk of various complications. These include nasal polyps, excessive mucus production, ear inflammation and pain, and sinusitis. Mucosal changes caused by the use of decongestants can disrupt the physiological functions of the nose related to temperature and humidity regulation and air filtration from contaminants [17]. As the disease progresses, atrophic changes in the nasal mucosa may predominate, including changes affecting the ciliary apparatus, and the mucosa may also become hardened. This can explain the occurrence of one of the rare secondary complications of RM, which is nasal septum perforation [18].

Diagnostics

No specific biochemical tests or imaging studies are used to confirm rhinitis medicamentosa.

Based on basic examinations, such as nasal endoscopy, it is not possible to differentiate RM from infectious or allergic rhinitis, as the appearance of the nasal mucosa is nonspecific [19]. During examination, the mucosa may appear swollen, often red (described as "beefy-red"), with pinpoint areas of bleeding and a small amount of mucus, although occasionally the mucus may be abundant [20]. In chronic cases, the nasal mucosa may become pale, atrophic, and hardened [21], so the appearance is not specific and can depend on both the duration of the disease process and individual characteristics.

Histological examination of the mucosa in guinea pigs revealed the presence of inflammatory cell infiltration, squamous metaplasia, goblet cell hypertrophy, and an increased number of glands [22].

There are also no specific criteria confirming RM. The diagnosis is based on physical examination and a thorough patient history, which includes typical symptoms and the use of described vasoconstrictor medications. The predominant reported symptom is the aforementioned nasal obstruction, which is subjectively assessed by the patient. However, additional, more complex tests can be performed, such as measuring nasal airway resistance (NAR), assessing nasal airflow, or nasal blood flow.

Importantly, other conditions that may present with similar symptoms must be excluded. This condition should be differentiated from allergic rhinitis, other types of non-allergic rhinitis, acute or chronic sinusitis, nasal polyposis, viral upper respiratory infections, or secondary rhinitis caused by a deviated septum or other obstruction [23][24].

Treatment

The primary step in managing rhinitis medicamentosa (RM) is to discontinue the use of nasal vasoconstrictor medications. Patients should be informed about the potential initial worsening of symptoms since the effects of the medication will wear off, likely increasing nasal obstruction. An inadequately educated patient might resort to using the medication again, creating a vicious cycle. Sometimes, it is recommended to continue using the vasoconstrictor in one nostril only to gradually reduce the dose, but this practice has not been confirmed in randomized studies [25]. It is also important to note that even short-term reuse of a nasal

vasoconstrictor, even a year after discontinuation, can cause a recurrence of RM [26]. Various treatments have been tried over the past few decades to alleviate rebound symptoms, including oral corticosteroids, antihistamines, and steroid injections into the inferior turbinates. Oral adenosine triphosphate and antihistamines have shown good efficacy, but the results are limited by small sample sizes and lack of similar conclusions [27].

Recent studies have increasingly focused on the effectiveness of nasal corticosteroids. Initial studies on animals induced RM by using a vasoconstrictor for 8 weeks, followed by treatment with fluticasone propionate nasal spray for 2 weeks [28], resulting in significant mucosal edema reduction. Similar results were observed in human studies, where patients using budesonide nasal spray for 6 weeks were able to discontinue vasoconstrictors without increased nasal obstruction [29].

Fluticasone propionate nasal spray was compared to placebo in RM treatment, showing faster and more effective results [30].

Despite numerous publications on RM treatment, a standardized treatment protocol has not been developed [31].

Combining nasal corticosteroids with the discontinuation of vasoconstrictors is the most commonly chosen treatment regimen, as confirmed by data from Canadian physicians [32].

Nasal corticosteroids are a common therapy for treating non-allergic rhinitis, with drug-induced rhinitis considered one of its forms [33]. However, according to a 2019 meta-analysis, the certainty of evidence for most outcomes related to this treatment was low or very low, and it was unclear whether nasal corticosteroids reduced the severity of patient-reported symptoms compared to placebo [34].

Treatment should last several weeks, depending on the rate of symptom resolution.

For persistent nasal obstruction, instead of resorting to vasoconstrictors again, hypertonic saline solution can be used. Although it has weak anti-edema effects, studies suggest it may have similar efficacy to vasoconstrictors in treating nasal obstruction.

An important aspect of RM treatment is facilitating the regeneration of the nasal mucosa. Nasal application of seawater plays a role in moisturizing and cleansing the mucosa.

Studies have shown that hypertonic seawater increases mucociliary clearance, which is comparable to the effect of oxymetazoline [35], highlighting the potential for gradually replacing vasoconstrictors with hypertonic saline solution while maintaining nasal obstruction improvement.

However, hypertonic saline, while enhancing mucociliary clearance, is more irritating than isotonic saline solutions, which have better moisturizing properties essential for mucosal regeneration [36].

Sea salt thus seems to be an important element in treatment when discontinuing decongestant drops; however, using it as a monotherapy may not be sufficient. According to an animal study, it was less effective than mometasone furoate in reducing mucosal swelling characteristic of RM [37]. Subsequently, if necessary, topical glucocorticoids (e.g. beclomethasone, flunisolide, budesonide, fluticasone, mometasone) can be used.

In one study, the effects of using hyaluronic acid intranasally twice daily for 10 days were compared with the use of a sea salt solution to discontinue decongestant medications. Both groups showed a significant reduction in mucosal swelling and nasal discharge, demonstrating the potential role of hyaluronic acid in this type of therapy; however, similar cases had not been documented [38].

If conservative treatment is insufficient and nasal obstruction severely affects the patient's quality of life or prevents discontinuation of vasoconstrictors, surgical methods may be considered.

A case was reported where a patient with RM, despite treatment with immunotherapy, antihistamines, and nasal steroid sprays, experienced significant nighttime breathing improvement only after conchoplasty. This combination of treatments rendered the patient asymptomatic [39].

Conchoplasty, often performed to reduce inferior turbinate swelling, includes laser conchoplasty, which is considered very safe with minimal complications, high precision, and

effectiveness [40]. In a study of 42 patients with refractory RM treated with laser conchoplasty, no postoperative complications were observed. It was assessed on visual analog scales (VAS) subjective severity of congestion and the patient's satisfaction before and after surgery. Visual analog scale (VAS) scores showed low pain and high patient satisfaction postoperatively, with significant improvements in objective rhinomanometry noted after half a year. A total of 88% of patients successfully stopped decongestant abuse after 6 months [41].

Another type of conchoplasty uses radiofrequency, which precisely reduces turbinate volume with minimal impact on surrounding tissues [42]. A questionnaire-based study described the impact of radiofrequency turbinate ablation on the quality of life in RM patients, indicating that conchoplasty improved nasal breathing and subjective quality of life [43]. Other described methods include cryosurgery and traditional mucotomy, involving surgical resection of turbinate fragments.

Conclusion

Nasal obstruction symptoms are frequently reported by patients, including adolescents and children. To obtain quick relief and eliminate the problem, these individuals often resort to widely available nasal decongestants. According to a survey conducted among doctors, warnings about the effects of these medications are inadequate, and they are not sufficiently visible to patients [44].

Lack of knowledge about the adverse effects resulting from prolonged use of these medications increasingly leads to addiction to these substances and consequently to the development of rhinitis medicamentosa and its associated chronic complications. This condition is not easy to treat, and therapy is often long and burdensome. It seems much easier to simply prevent the development of RM by avoiding such medications or using decongestant nasal drops only for short periods and in rational doses. Specialists in otorhinolaryngology, family medicine, pediatrics, and other specialties, as well as pharmacists, should emphasize the dangers of these medications. This is particularly necessary because these drugs are usually available over the counter and are heavily advertised (especially on television), which further encourages their use.

These conclusions were included in a 2009 study [45], which suggested that the intensive media advertising campaign for topical decongestants that began a few years earlier was correlated with an increase in RM after a few years. Therefore, it is crucial to promote information among patients about the adverse effects concerning prolonged and improper use of popular and widely available nasal decongestants, as this can prevent many individuals from developing rhinitis medicamentosa.

DISCLOSURES

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