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REVIEW ARTICLE

Rebound congestion – a diagnostic and therapeutic challenge for the family physician and laryngologist

Katar z odbicia – wyzwanie diagnostyczno-terapeutyczne dla lekarza rodzinnego i laryngologa

Authors' Contribution:
A – Study Design
B – Data Collection
C – Statistical Analysis
D – Data Interpretation
E – Manuscript Preparation
F – Literature Search
G – Funds Collection

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ABSTRACT: Rhinitis medicamentosa (RM), also known as "rebound congestion" or drug-induced rhinitis, is a chronic condition of the nasal mucosa resulting from long-term use of intranasal decongestants. The disorder poses a significant clinical problem due to the high availability of OTC sympathomimetics and the lack of patient awareness regarding the risks of their abuse. The pathophysiology of RM has not been fully elucidated. Clinically, the disorder manifests itself through chronic nasal congestion, frequently accompanied by sleep disturbances, snoring, and decreased quality of life. Diagnosis is based mainly on the history of the use of decongestants and the exclusion of other causes of nasal obstruction. The mainstay of treatment consists in immediate or gradual discontinuation of the sympathicomimetic supported by intranasal corticosteroids, saline or hypertonic saline solutions, and mucosa-regenerating preparations such as dexpanthenol. In selected cases, a short course of oral steroids can be used, or surgical treatment can be considered, particularly upon coexisting anatomical abnormalities. Patient education and proper recommendations regarding short-term use of decongestants and alternative therapies play a key role in the prevention of RM. Early recognition and appropriate treatment facilitate the avoidance of chronic complications and disease recurrence.

KEYWORDS: alpha-mimetics, dexpanthenol, rebound congestion

STRESZCZENIE: *Rhinitis medicamentosa* (RM), określana jako „katar z odbicia" lub polekowy nieżyt nosa, jest przewlekłym schorzeniem błony śluzowej nosa wynikającym z długotrwałego stosowania donosowych leków obkurczających. Choroba stanowi istotny problem kliniczny ze względu na dużą dostępność sympatykomimetyków OTC oraz brak świadomości pacjentów dotyczącej ryzyka ich nadużywania. Patofizjologia RM pozostaje nie w pełni poznana. Klinicznie RM manifestuje się przewlekłą niedrożnością nosa, często z towarzyszącymi zaburzeniami snu, chrapaniem i pogorszeniem jakości życia. Diagnostyka opiera się głównie na wywiadzie dotyczącym stosowania leków obkurczających oraz wykluczeniu innych przyczyn niedrożności nosa. Podstawą leczenia jest natychmiastowe lub stopniowe odstawienie sympatykomimetyku, wspomagane terapią donosowymi glikokortykosteroidami, roztworami soli fizjologicznej lub hipertonicznej oraz preparatami regenerującymi błonę śluzową, takimi jak deksopantenol. W wybranych przypadkach stosuje się krótki kurs steroidów doustnych lub rozważa leczenie chirurgiczne, zwłaszcza przy współistniejących zaburzeniach anatomicznych. Kluczową rolę w profilaktyce RM odgrywa edukacja pacjentów i właściwe zalecenia dotyczące krótkotrwałego stosowania leków obkurczających oraz alternatywnych metod terapii. Wczesne rozpoznanie i odpowiednie leczenie pozwalają uniknąć przewlekłych powikłań oraz nawrotów choroby.

SŁOWA KLUCZOWE: alfa-mimetyki, dekspantenol, katar z odbicia

ABBREVIATIONS

AR – allergic rhinitis
EGF – epidermal growth factor
ENT – Ear, Nose, and Throat
OTC – over-the-counter
RM – rhinitis medicamentosa

INTRODUCTION

Rhinitis medicamentosa (RM), known as "rebound congestion" or drug-induced rhinitis, is a form of inflammation of the nasal mucosa resulting from long-term use of intranasal decongestants (sympathomimetics) [1]. It is defined as persistent nasal obstruction without concomitant rhinorrhea or postnasal drip, developing after excessive use of intranasal decongestants (usually for more than 7–10 days). Symptoms can manifest both shortly after withdrawal (as early as about 3 days) or over a more distant period up to 4–6 weeks [2]. It is most common in young and middle-aged individuals, with similar frequency in men and women. According to various estimates, the condition occurs in 1–9% of outpatients presenting at the ENT clinics [3].

The pathophysiology of RM has not been clearly elucidated, with several potential mechanisms for its development having been proposed in the literature:

- hypothesis 1: long-term vasoconstriction leads to mucosal ischemia, which promotes interstitial edema;
- hypothesis 2: sustained stimulation of vasoconstrictive mechanisms causes their gradual "exhaustion", resulting in reactive hyperemia and increased congestion. As a result, sensitivity to endogenous catecholamines is reduced, and the response of adrenergic receptors to the decongestant agents is weakened, necessitating further increase in administered doses (tachyphylaxis);
- hypothesis 3: changes in the regulation of vascular tone result in an increase in vascular wall permeability, exacerbating edema;
- hypothesis 4: prolonged activity of β-adrenergic receptors relative to α-adrenergic receptors can induce secondary vasodilatation, leading to the so-called rebound effect [2].

There is a number of factors that predispose patients to the development of this condition. The most important of these include:

- chronic use of intranasal decongestants, especially beyond the recommended time (usually > 5–10 days). Xylometazoline and oxymetazoline, as α-adrenergic receptor agonists, induce vasoconstriction of mucosal blood vessels, leading to a reduction in congestion, exudate, and tissue edema. As a result, local inflammatory reaction is alleviated, and nasal patency and sinus drainage are improved. *In vitro* studies showed that oxymetazoline produced a stronger and more prolonged effect (up to 12 hours) compared to xylometazoline (up to 10 hours);
- the ease of access to over-the-counter (OTC) preparations and insufficient information about possible negative effects on labels;
- lack of adequate education on the use of preparations;
- patients' failure to comply with recommendations regarding the use period;
- diseases that promote prolonged use of decongestants, e.g., chronic sinusitis;
- behavioral and psychosocial features, such as addictive use of nasal sprays, depressive disorders, or a vicious cycle mechanism where chronic use of decongestants intensifies the feeling of nasal congestion, thus forcing continued use and the need to increase doses [4–6, 7, 8].

CLINICAL PRESENTATION

The clinical presentation consists in the sensation of a chronically congested nose with swelling within the nasal cavity, leading to difficulties in fully taking in air. Clinical symptoms may also include snoring and insomnia resulting from upper airway obstruction and breathing through the mouth. Rhinoscopic presentation does not allow clear differentiation between RM and infectious or allergic rhinitis. The mucosa may be beefy red with pinpoint petechiae and trace amounts of discharge, albeit significant swelling with profuse, sticky, mucous discharge is equally common. In cases of chronic abuse of sympathomimetic, the mucosa can become pale and swollen or, in advanced stages, atrophic and scabby [9, 10].

Chronic inflammatory and structural changes induced by decongestants impair the key nasal functions, such as filtration or regulation of temperature and humidity of the inhaled air. Consequently, the condition can predispose those affected to the development of complications, including chronic sinusitis, otitis media, nasal polyps, or atrophic rhinitis [9, 10].

DIAGNOSTICS

The diagnosis of RM is made mainly on the basis of history and clinical presentation. The first diagnostic criteria, as proposed in 1952, included:

1. prolonged use of intranasal drugs;
2. permanent nasal obstruction;
3. absence of mucosal decongestion upon physical examination [11].

Despite the passage of years and numerous other parameters reflecting the nature of the pathology being postulated mostly on the basis of retrospective studies, clear and validated criteria for diagnosis are still lacking [9].

The main feature of drug-induced rhinitis is, by definition, the history of the use of drugs, primarily topical nasal mucosal decongestants. The duration of decongestant treatment required to result in RM is widely debated and has not been clearly defined; according to some authors, rebound congestion does not develop before 8 weeks of topical mucosal decongestant treatment [12, 13] while others suggest that the onset of the disease may occur after as few as 3–10 days of use [14, 15].

Nasal obstruction characteristic of RM is a nonspecific symptom, and other disorders involving sinonasal mucosa must coexist at presentation or in the recent history of patients with the suspicion of

RM. Interviewing the patient is one of the most important elements of the diagnostic process. A thorough history facilitates not only the preliminary diagnosis, but also identification of overlapping sources of nasal obstruction or inflammation such as allergic rhinitis, other forms of non-allergic rhinitis, or rhinosinusitis (Tab. I.) [16].

Alarming symptoms include unilateral discomfort (raising the suspicion of a foreign body in children or a tumor in adults), recurrent nosebleeds or mucopurulent or bloody nasal discharges, pain complaints, or olfactory disturbances [18].

Subjective examination should be complemented by a physical examination: anterior rhinoscopy, and preferably – and unconditionally in all patients with alarming symptoms – endoscopic examination of the nasal and nasopharyngeal cavities.

The main disorders to be taken into account in differential diagnosis of nasal patency disorders include nasal septum deviation, sinonasal polyps and other rhinosinusitides, hypertrophy of the pharyngeal tonsil or nasopharyngeal tumor, tumors of the maxillofacial massif as well as disorders of ciliary structure or function (e.g., cystic fibrosis) or other diseases manifesting within the upper respiratory tract (e.g., granulomatosis with polyangiitis) (Tab. II.) [18].

The presentation of nasal mucosa in drug-induced rhinitis is non-specific and resemblant of allergic, infectious, or atrophic rhinitis. Redness and swelling, as well as irregular surface and cobblestone texture of the mucosa can be observed along with the absence of reaction to mucosal decongestants. The risk of vasoconstriction-related ischemia of the nasal septum mucosa resulting in mucosal drying and scabbing, and ultimately perforation of the nasal septum, must also be kept in mind.

In order to understand the nature of the pathology, as well as to identify possible underlying causes of nasal patency disorders, particularly in refractory cases, consideration should be given to expanding the diagnostic workup to include allergy screenings and cytological studies [19]. Histological examination of the mucosa reveals an absence of cilia, squamous cell metaplasia, goblet cell hyperplasia, increased number of submucosal glands, increased expression of epidermal growth factor (EGF) receptors, and infiltration with inflammatory cells, mostly neutrophils [19, 20].

No imaging studies are usually performed in the absence of the alarming symptoms or rhinoscopic/endoscopic presentation; in doubtful cases, computed tomography is the imaging modality of choice [4].

Rhinomanometry is a useful tool for objective examination of nasal patency by analyzing nasal flow and resistance. Acoustic rhinometry, which evaluates changes in the geometry of the airways, can be applied for a similar purpose. It is considered worthwhile to use both of these methods as complementary diagnostic tools to facilitate standardization of treatment results as the methods are usually pursued if surgical treatment is planned [16].

Tab. I. Symptoms of allergic rhinitis [17].

SYMPTOMS INDICATIVE OF AR	SYMPTOMS USUALLY UNRELATED TO AR
2 or more of the following symptoms lasting >1 h on most days	
Watery nasal discharge	Unilateral symptoms
Sneezing, especially paroxysmal	Mucopurulent nasal discharge
Impaired nasal patency	Facial/nose pain
Nasal pruritus	Recurrent nosebleeds
Conjunctivitis	Olfactory impairment
	Postnasal drip (thick mucus)
	Isolated nasal discharge

AR – allergic rhinitis

Tab. II. Differential diagnosis of nasal patency disorders.

Nasal septum deviation
Hypertrophy of the inferior nasal turbinates
Anatomical abnormalities of the lateral wall of the nasal cavity (e.g., aerated middle nasal turbinate).
Chronic rhinosinusitis with or without nasal polyps
Hypertrophy of the pharyngeal tonsil
Nasal tumors
Foreign body in the nose
Disorders of ciliary function or structure (cystic fibrosis, primary ciliary dyskinesia)
Other diseases manifesting in the upper respiratory tract (e.g., sarcoidosis, granulomatosis with polyangiitis, eosinophilic granulomatosis with polyangiitis)

TREATMENT

Although rebound congestion has been recognized as a clinical entity for several decades, a widely accepted treatment protocol aimed at weaning off decongestants has not been developed yet [21]. The lack of a standard treatment regimen is directly related to the uncertainty regarding the pathophysiological mechanism underlying RM as reflected in the available literature.

The primary element of management consists in discontinuation of topical nasal mucosal decongestants [16]. By discontinuing the use of intranasal alpha-mimetics, nasal function can be normalized, and symptoms can be resolved. Patients should be warned in advance that nasal patency would temporarily deteriorate; otherwise, they may resume the use of topical nasal mucosal decongestants within a few days. Both abrupt and gradual withdrawal of the drug is acceptable [1]. Gradual reduction may involve a reduction in concentration or frequency of use as well as initial cessation of use in one nasal cavity, allowing the patient to maintain satisfactory patency until stabilization of the condition of the mucosa on the contralateral side, where the drug has been discontinued [22].

The main role in relieving the symptoms of recurrent mucosal edema is played by nasal corticosteroids [15, 23]. The choice of the type of topical corticosteroid is not important, as there is no difference in the clinical efficacy of different types of preparations [24]. For maximum effect, corticosteroids are recommended for a period of up to six--eight weeks [1, 15], and dosing usually involves administration of

up to 2 doses into both nasal cavities twice a day [25]. The findings of a systematic review covering the outcomes of RM indicate that topical steroids such as budesonide, fluticasone propionate, and dexamethasone are effective in relieving the symptoms of rebound congestion [26]. Studies evaluating the effects of mometasone furoate in experimental animal models of RM have also shown a reduction in swelling and mucosal regeneration [26, 27].

Complementary therapeutic options alternative to nasal mucosal decongestants can also be suggested, such as intranasal physiological saline [16] or, particularly in the initial period, hypertonic saline [19]. Nasal lavage using saline or seawater solutions helps clear nasal passages, improve mucociliary transport, and maintain hydration. In addition, hypertonic saline solution has a weak antiedemic effect, its clinical value being demonstrated by Garavello et al. in a series of patients treated with 3.0% hypertonic saline solution three times a day during the pollen season [28] as well as by Rabago et al. in a series of patients treated with hypertonic saline solution compared to patients using isotonic saline solution [29]. The use of hypertonic solutions may be associated with mild adverse effects such as irritation of the nasal mucosa and a stronger burning sensation; however, such effects are rare, and serious side effects are virtually nonexistent [30].

Additional ingredients, such as dexpanthenol or hyaluronic acid, can further promote mucosal healing and hydration. Dexpanthenol has been shown to strengthen the epithelial barrier, prevent irritation, and stimulate mucosal regeneration [31]. Another supporting factor is the hyaluronic acid; contrary to some emerging opinions, it has no direct regenerative properties similar to those of dexpanthenol, while its moisturizing effect should certainly be noted. It is therefore worth looking at combinations of hypertonic salts, for example with dexpanthenol, available in the form of intranasal sprays. This form of administration may be strongly preferred, especially for those with poor tolerance of sinus lavage. Other drugs used in the treatment of rebound congestion include oral steroids (e.g., prednisone 0.5 mg/kg for 5 days or 3 × 15 mg for 5 days [2, 25], oral nasal mucosal decongestants (after the risk of side effects is taken into account), topical or oral antihistamines (if AR had been originally present) and mast cell stabilizing drugs. The level of evidence for the effectiveness of these treatments is limited to case reports or case series [9, 25].

In refractory cases, surgical intervention should be considered. Structural abnormalities such as a deformed nasal septum, nasal polyps, or anatomical variations of the lateral wall of the nasal cavity, potentially contributing to prolonged use of mucosal decongestants, are indications for surgical treatment. Persistent hyperplasia of inferior nasal turbinates unresponsive to conservative treatment, along with the associated nasal patency disorders, should prompt consideration of conchoplasty. Both submucosal resection techniques and radiofrequency or laser conchoplasty have been proposed for the treatment of recurrent RM [1, 20, 23, 32]; posterior nasal nerve neurolysis, as performed concurrently with conchoplasty, has also shown promising results in patients with comorbid symptoms such as rhinitis, pruritus, and sneezing [33]. At the same time, however, it is believed that intranasal procedures carry an additional risk of damage to the nasal mucosa, and therefore, at least 3 months of successful RM treatment (i.e., no need for decongestants) are advocated before considering any intranasal surgical intervention. These conflicting perspectives underscore the need for further research to clarify the indications and long-term outcomes of surgical treatment for RM [24]. A proposed management algorithm is shown in Fig. 1.

PATIENT EDUCATION, OR HOW TO REDUCE THE RISK OF DEVELOPING RHINITIS MEDICAMENTOSA

The best solution to the problem of rebound congestion is to prevent its occurrence. This can be achieved through proper communication, hence the importance of educating patients at risk of the disease. Patients just starting therapy with sympathomimetics should be warned of the risk of addiction and paradoxical effects of the drug upon longer-than-recommended use. Neither the cumulative dose of nasal mucosal decongestants nor the exposure period necessary to induce RM has been clearly defined to date, and therefore it is critical to advise patients that the treatment be continued for the shortest necessary period [35].

Because of the widespread, easy access to α-sympathomimetics, pharmacists also play an extremely important role in patient education, reminding patients of the risks of addiction and rebound congestion.

When recommending intranasal preparations, one may consider an alpha-mimetic containing additional ingredients with moisturizing or regenerative effects, such as dexpanthenol or hyaluronic acid, so as to support the regeneration of nasal mucosa, moisturizing it and protecting its epithelium from further damage caused by the ongoing inflammatory process. It is worth bearing in mind that the likelihood of overuse of decongestant drops decreases significantly among those treated with concomitant intranasal steroids and those using other adjunctive medications, such as oral antihistamines, oral mucosal decongestants, or saline solutions [36]. The possibility of using an additional intranasal preparation like hypertonic saline not only after the planned treatment with a sympathomimetic, but also alternating therewith in the first phase of the treatment, positively influences compliance and reduces the risk of prolonging treatment with the mucosal decongestant.

Anxiety, sleep disorders, smoking, diseases requiring opioids, and local inflammation of the nasal mucosa, as assessed by cytologic examination, were identified as factors particularly predisposing patients to the abuse of topical nasal mucosal decongestants [19]. The aforementioned risk factors should be considered when recommending treatment for nasal obstruction. Adjunctive preparations should be particularly considered in this group of patients.

CONCLUSIONS

RM is a chronic condition, widely encountered in the daily medical practice of not only otorhinolaryngologists but also primary care physicians. Changes within the mucosa of the nasal cavity resulting

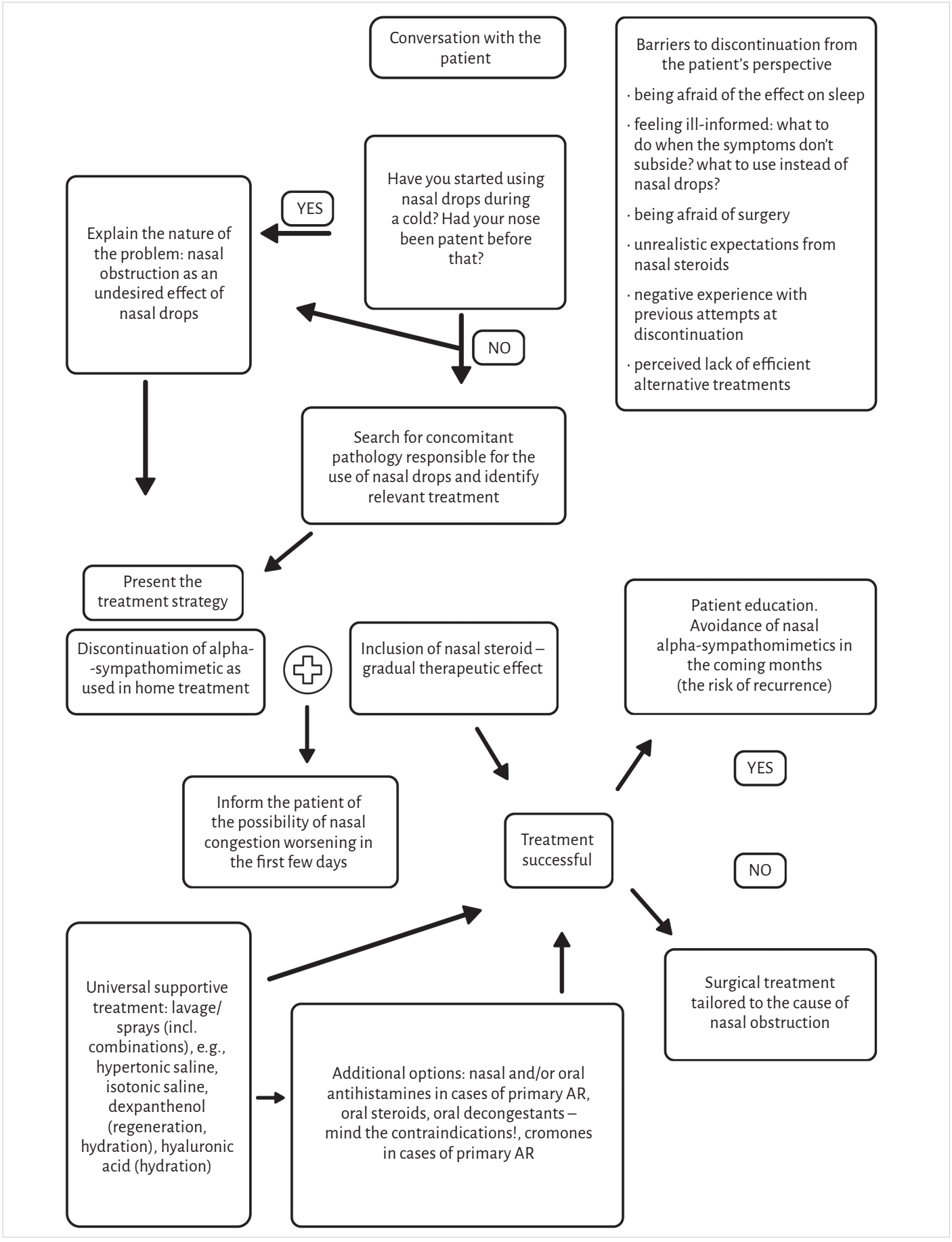


Fig. 1. The proposed management regimen for RM [34].

from the excessively long use of decongestants can lead to chronic nasal obstruction. Thus, a key role in reducing the incidence of RM is played by consistent education of patients as provided by physicians and pharmacists, including education on proper use of medications and information about safe alternatives. In addition, early diagnosis and initiation of effective treatment reduce the risk of long-term complications and disease recurrence. In the event of RM, therapeutic intervention is needed as soon as possible, including discontinuation of current decongestants and implementation of dedicated treatment, which may include intranasal steroid therapy, isotonic/hypertonic saline solutions preferably with adjunctive agents such as dexpanthenol, and oral preparations.

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