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ROLES OF FAMILY DOCTORS IN MANAGING ALLERGIC RHINITIS

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Abstract:

Allergic rhinitis and asthma are very widespread around the world. Recognizing the pathophysiology would cause an improved medical management of these diseases. In this review we discuss the background and management of Allergic rhinitis. We conducted a literature review of articles published up to 2018, in following databases; PubMed, and Embase overviewing the allergic rhinitis management in family medicine by family physicians. AR is worth dealing with efficiently, even when it is part of a myriad of allergic conditions; as the nose is the gateway to the respiratory system tract, good rhinitis control can facilitate control of signs in other places. AR, if badly managed, results in bothersome signs and effect on daily activities, quality of life and on other areas of the respiratory tract, such as ears, sinuses, throat and lungs. Feasible reasons for difficult-to-treat instances consist of physician factors such as misdiagnosis and under treatment or patient aspects such as insufficiency of concordance with treatment. This can be decreased by information brochures on specific irritant evasion, the proper strategy in the application of nasal preparations and explanation of prospective adverse effects. Clearly written therapy strategies work, particularly if a number of preparations are suggested.

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INTRODUCTION:

Rhinitis is generally specified as inflammation of the nasal mucosa. It is a common disease that influences approximately 40% of the populace [1]. Allergic rhinitis is one of the most prevalent sort of chronic rhinitis, impacting 10 to 20% of the populace, and proof proposes that the prevalence of the ailment is intensifying. Serious allergic rhinitis has actually been connected with considerable disabilities in quality of life, sleep and work performance [1]. The symptoms of AR, rhinorrhea, nasal obstacle, itching, and sneezing, are in an instant brought on by exposure to irritants and inducing aspects, and may be reversible [1]. AR is a major airway illness, which causes morbidity, health care expenditures, and dramatically weakens a patient's ability to function and their quality of lifestyle. It is also co-morbid with asthma, sinusitis, anosmia, otitis media, nasal polyps, lower airway infections, and dental malocclusion [1].

For patients experiencing AR, general practitioners are commonly their first source of clinical recommendations [4]. It is one of the top-ten causes for a visit to the medical care clinics and AR was estimated to be 10-40% of the total patient check outs in concerning 50% of the primary care facilities [2]. It has been reported in a population-based survey research that 71% of the rhinitis patients went to a health care doctor and just 18% an Otolaryngologist (ENT specialist) in Singapore [3].

As lots of rhinitis patients depend on their general practitioners (GPs) for the diagnosis and treatment of their signs, general health care methods stand for an interesting and important target to be examined as part of the management of AR [2]. Throughout the past couple of years, international standards and agreement statements for the management of AR have been created to improve the performance and quality of management for AR patients [1].

Allergic rhinitis and asthma are very widespread around the world. Recognizing the pathophysiology would cause an improved medical management of these diseases. In this review we discuss the background and management of Allergic rhinitis.

METHODOLOGY:

We conducted a literature review of articles published up to 2018, in following databases; PubMed, and Embase overviewing the allergic rhinitis management in family medicine by family physicians. We restricted our search to only English published articles with human subjects concerning. More studies were recruited from scanning the bibliography of found studies to have more support evidence.

DISCUSSION:

- **CLASSIFICATION OF RHINITIS**

Rhinitis is identified into one of the following classifications according to etiology: IgE-mediated (allergic), autonomic, infectious and idiopathic (unidentified). Although the focus of this article is allergic rhinitis, a short description of the various other types of rhinitis is given in Table 1.

Table 1. Etiological classification of rhinitis [1].

	Description
IgE-mediated (allergic)	• IgE-mediated inflammation of the nasal mucosa, resulting in eosinophilic and Th2-cell infiltration of the nasal lining • Further classified as intermittent or persistent
Autonomic	• Drug-induced (rhinitis medicamentosa) • Hypothyroidism • Hormonal • Non-allergic rhinitis with eosinophilia syndrome (NARES)
Infectious	• Precipitated by viral (most common), bacterial, or fungal infection
Idiopathic	• Etiology cannot be determined

Generally, allergic rhinitis has been classified as seasonal (takes place throughout a certain period) or perennial (takes place throughout the year). However, not all patients fit into this category scheme. For instance, some allergic triggers, such as pollen, may be seasonal in cooler environments, although perennial in warmer environments, and individuals with multiple "seasonal" allergies may have symptoms throughout a lot of the year [4]. As a result, allergic rhinitis is now classified according to sign duration (intermittent or persistent) and seriousness (mild, moderate or severe) (see Figure 1) [1]. Rhinitis is considered intermittent when the total period of the episode of inflammation is less than 6 weeks, and consistent when symptoms continue throughout the year. Signs are classified as mild when patients are typically able to sleep normally and perform typical activities (consisting of work or school); mild symptoms are usually recurring. Symptoms are categorized as moderate/severe if they substantially influence sleep and activities of everyday living and/or if they are taken into consideration bothersome. It is very important to classify the seriousness and duration of signs as this will direct the management strategy for private patients [1].

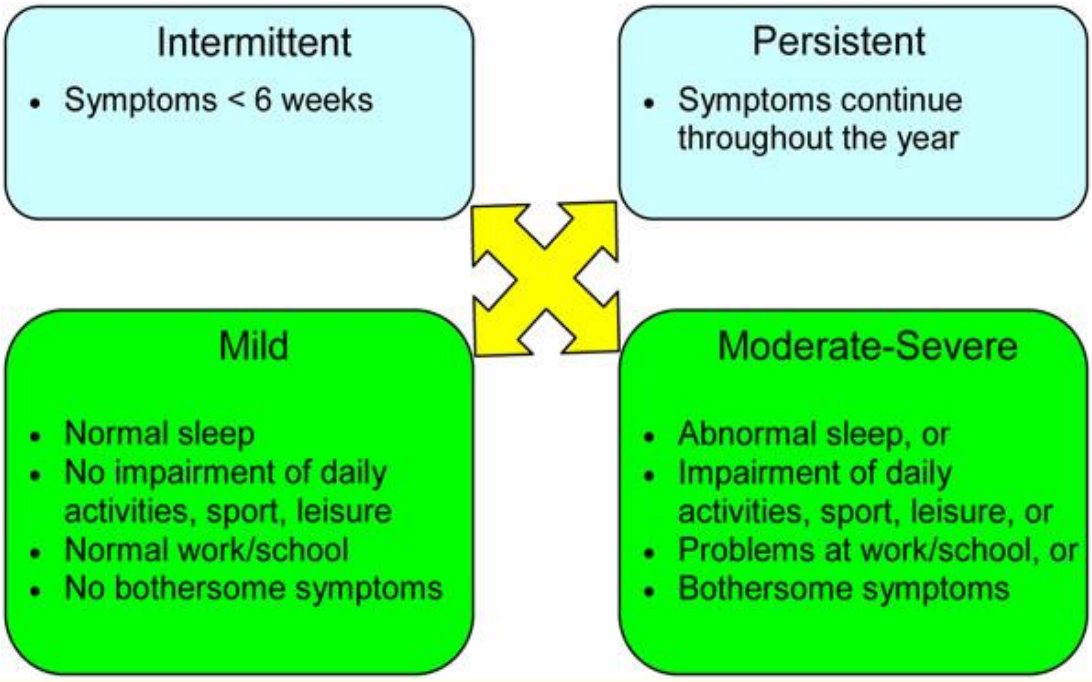

Figure 1. Classification of allergic rhinitis according to symptom duration and severity [1].

- **PATHOPHYSIOLOGY OF AR**

Sensitization to allergens

Antigen presenting cells (APCs), such as dendritic cells in the mucosal surface, procedure allergens and offer some peptides from allergens on the major histocompatibility complex (MHC) course II particle [5]. This MHC class II molecule and antigen complex take a function as the ligand of T-cell receptors on Naive CD4+T cells, which result in distinction of Naive CD4+T cells to allergen-specific Th2 cell. Turned on Th2 cells secret a number of cytokines, which induce isotype changing of B cells to create certain IgE and proliferation of eosinophils, mast cells and neutrophils (figure2) [6]. Generated antigen-specific IgE binds to high-affinity IgE receptors on mast cells or basophils.

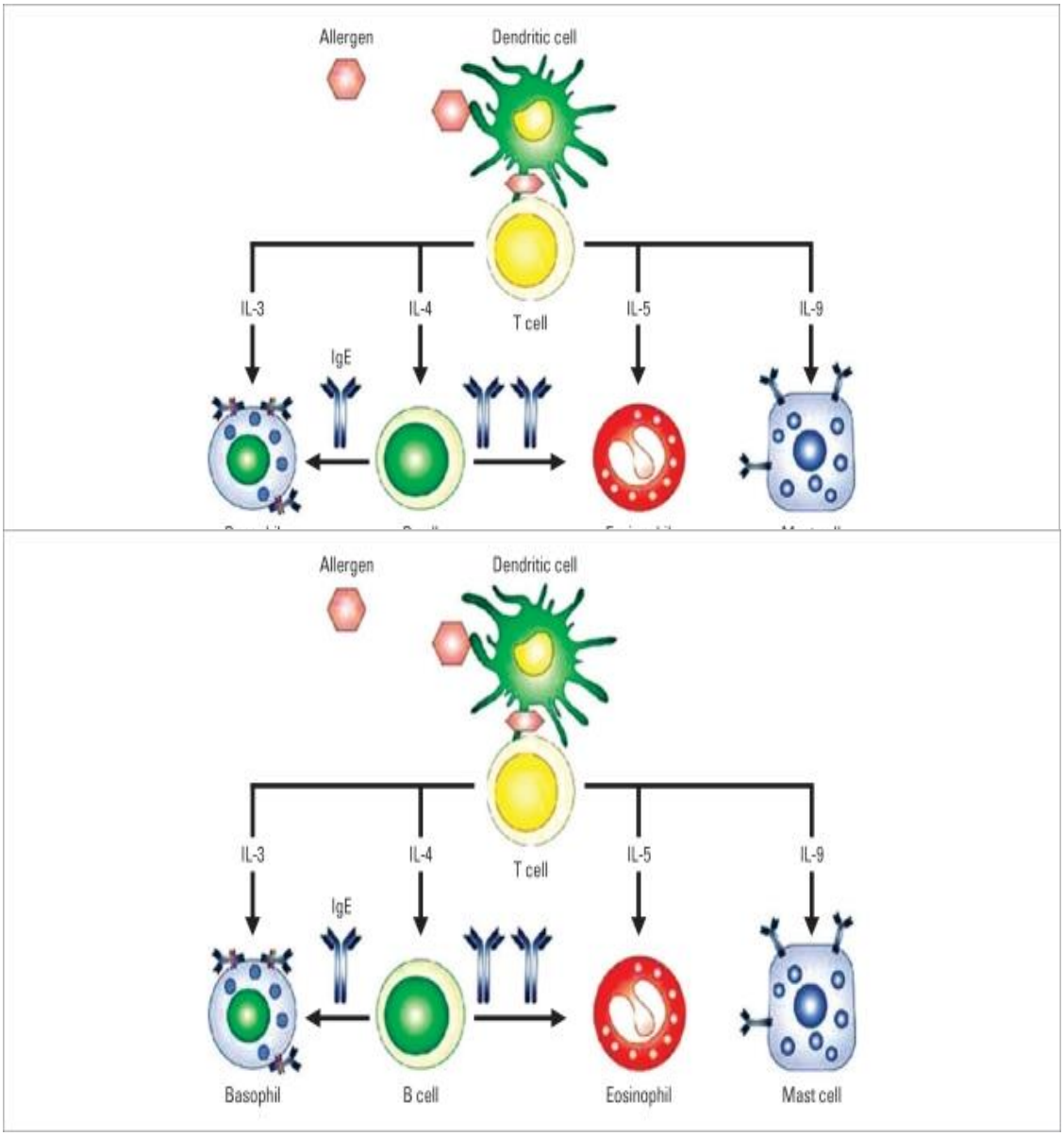

Figure 2. Allergen-induced sensitization and inflammation [6].

Early and late reactions

When AR patients are exposed to allergens, allergic reactions establish in 2 various patterns according to time series. One is the early reaction, in which sneezing and rhinorrhea establishes in 30 minutes and vanishes. The other is the late reaction, which shows nasal obstacle about 6 hours after exposure to allergens and subsides gradually. The early reaction is the response of mast cells to offending irritants (type I hypersensitivity). Stimulated mast cells induce nasal symptoms by secreting chemical mediators such as histamine, prostaglandins and leukotrienes [7]. Unlike the early response, eosinophil chemotaxis is the main system in the late reaction, which is brought on by chemical mediators created in the early reaction. Several inflammatory cells, eosinophils, mast cells and T cells move to nasal mucosa, break up and remake regular nasal tissue, and these processes lead to nasal obstruction which is the main symptom of AR patients [8].

Neurogenic inflammation

When breathing epithelium is destroyed and nerve endings are exposed by cytotoxic proteins from eosinophils, sensory nerve fibers are excited by nonspecific stimuli and stimulate both sensory afferent and surrounding efferent fibers, the socalled retrograde axonal response. This makes the sensory

nerve fibers secrete neuropeptides such as compound P and neurokinin A, which induce tightening of smooth muscle mass, mucous secretion of goblet cells and plasma exudation from capillaries. This procedure is called neurogenic inflammation [9].

Non-specific hyperresponsiveness

Non-specific hyperresponsiveness is just one of the scientific characteristics of allergic inflammation. Due to eosinophilic infiltration and damage of nasal mucosa, the mucosa becomes hyperactive to regular stimuli and causes nasal signs such as sneezing, rhinorrhea, nasal itching and obstruction [10]. This is a non-immune reaction that is not associated with IgE. Hypersensitivity to non-specific stimulations such as tobacco or cold and dry air as well as particular allergens boosts in AR patients.

Relationship between AR and asthma - "One airway, one disease"

Epidemiologic studies have declared that almost all of the asthma patients have AR symptoms eosinophilic inflammation of nasal mucosa despite nasal symptoms [11]. This characteristic finding of asthma is not found in patients with other pulmonary ailments and is the evidence that asthma is a systemic illness. The prevalence of asthma in AR patients has been reported to be from 10% as much as 40% [11]. A number of clinical studies have actually revealed that although AR patients do not have asthma, they can have eosinophilic infiltration in bronchial mucosa [11]. Madonini et al. have suggested that AR patients with positivity to pollens have bronchial hyperresponsiveness throughout the pollen period [12]. Corren et al. have demonstrated that irritant stimulation to nasal mucosa in AR patients causes bronchial hyperresponsiveness [13]. With collective of evidence for structural analog and the similarity of allergic inflammatory cells, inflammatory mediators and cytokines between top and lower air passages, the "one airway, one illness" idea has actually been presented [14]. This indicates that ever since AR and asthma are not different illness entity, simultaneous asthma in AR patients and simultaneous AR in asthmatic patients need to be indentified and both upper and lower airway allergy should be treated all at once. The ARIA (2008) advises that asthma must be reviewed in moderate-severe persistent AR patients.

- **MANAGEMENT OF AR PATIENTS IN PRIMARY CARE PRACTICE**

In the United Kingdom, a national standard audit of GPs with a self-declared interest in allergic and respiratory system disorders exposed substantial extent for improvement in GP recognition and management of AR [16]. In this research study, just 14% of GPs pleased all the standards set for identification of symptoms; 23% satisfied requirements for collection of information to support a medical diagnosis; 0% satisfied criteria for evaluation and examinations performed to support the medical diagnosis; and 0.6% satisfied requirements set for sufficient treatment released. In France, it was shown that the habits of medical techniques (by GPs) are usually, but not constantly, regular with one of the most current international agreement documents [18].

A conventional diagnostic approach recommended by ARIA is a careful history (Table 2), a nasal assessment and allergy tests (skin tests, in vitro tests or even nasal obstacle) to confirm or omit an allergic etiology [19]. Nonetheless, because of the deficiency of technological support and manpower in the majority of GPs' clinics, allergic reaction examinations are not typically done (around 50%) by GPs [20]. In addition, it has been argued that the typical nasal allergies can be diagnosed with a careful research study of symptoms and the response to preliminary therapy [17]. This is a crucial controversy that will impact substantially the standardization of allergy diagnosis. It is not possible to set apart the sort of rhinitis (infectious, allergic or other origins) based solely on signs and symptom measures, especially for persistent allergic rhinitis (PER). For that reason, an agreement has to be made by multidisciplinary clinical organizations (e.g., allergology, otolaryngology and GPs) in order to avoid an irregular or incorrect medical diagnosis of AR.

The standard of management for AR amongst GPs is thus an essential end result assessment for implementation of global guidelines. In Belgium, a study was able to reveal that when contrasted purely with the ARIA referrals, 49% of the patients with mild and/or intermittent AR were over-treated, whereas about 30% of those with moderate/severe consistent rhinitis were under-treated [20]. This research suggests that additional initiatives are called for to share and carry out evidence-based analysis and therapy guidelines for AR in primary heathcare method.

The GPs considered perennial allergic rhinitis (PAR) to be harder to deal with than seasonal allergic rhinitis, and GP and patient level of contentment in the treatment of PAR was reduced [21]. A similar outcome was experienced in a populace research study in Singapore where PER is nearly solely the pattern of AR seen, due to a typical tropical

environment and high interior degree of apartment dust mite irritants [15]. The effectiveness of treatment was usually thought about unsuitable by the patients since the large number had just partial or no relief of their signs. Patient with PER will require treatment the year around which may impact the selection of treatment and cost of treatment. Most patients anticipate quick symptomatic alleviation with affordable medicines.

In several countries, GPs are anticipated by the insurance companies and patients to supply both an office browse through and drugs for a nominal cost. Therefore, physicians must see a large number of patients to fulfill their expenses and do not take time to notify individual patients regarding their illness or to provide lengthy instructions on exactly how to use medications appropriately. A study carried out in the Netherlands showed that GPs seem likely to avoid co-payment for patients when these patients have economic difficulties and the condition is perceived as severe [22]. They selected totally compensated medicines. This might partly describe why an unanticipated low use nasal glucocorticosteroids and more recent generation antihistamines, and an usual use of nasal/oral decongestants in order to give fast symptomatic alleviation of nasal clog.

Single or combined therapy utilizing newer generation antihistamines and nasal glucocorticosteroids are suggested as conventional pharmacologic agents in the treatment of AR [19]. In practice this idea has actually not always been approved worldwide. It has been reported that only 45% of patients are treated with nasal glucocorticosteroids, contrasted to more than 90% with oral antihistamines [23]. In this research, nearly all GPs (99.5%) showed that they would certainly recommend H1-antihistamines (especially very first generation) and nasal glucocorticosteroids (95.5%) in the therapy of AR. Our previous study showed an unanticipated reduced use nasal glucocorticosteroids sprays (3%) and antihistamines (6%) in area AR patients in Singapore [25]. In one more research study in Singapore, information shows that the majority of GPs were up to-date with current progression in clinical allergic reaction and pharmacologic research. They understood rather well the efficiency, adverse effects and cost efficiency of 1st and latest generations of H1-antihistamines and nasal glucocorticsteroids. However, the cost of the new H1-antihistamines can be over 100 times more than the initial generation H1-antihistamines. Nasal glucocorticosteroids sprays are additionally costly, which is a crucial problem for patients with relentless (or perennial) AR that require long-term medication. It is therefore important that the proper use nasal steroid sprays and the beginning of medical efficiency need to be completely described to the patients.

In Singapore, PER is nearly exclusively the pattern of AR seen, as a result of a normal exotic climate which is hot and moist throughout the entire year. The year-round warm and moist environment contributes for the expansion of dust mites and molds, two of one of the most common aeroallergens linked in PAR. House dust mites are the most common recognized indoor allergens, and add to the advancement of AR and asthma. International researches have shown that Dermatophagoides pteronyssinus, Dermatophagoides farinae, and Euroglyphus maynei are the most common mite types globally [1]. These mites feed on human skin dander, and are specifically abundant in mattresses, pillows, and carpetings. Their growth is optimum under hot (above 20%) and damp conditions (80% relative humidity) [24].

Patients with PER will need therapy the year around which may affect the selection of therapy and price of therapy. Most patients anticipate fast symptomatic relief with inexpensive drugs. In Singapore, along with in lots of various other countries, GPs are expected by the insurance provider and patients to give both an office go to and drugs for one small cost. As repercussion physicians should see a great deal of patients to meet their expenses and do not take time to educate individual patients about their illness or to offer extensive guidelines on how to make use of medicines effectively. It might also describe why nasal decongestants are frequently made use of in order to provide fast relief of nasal obstruction. However, the extended use topical nasal decongestants can actually be hazardous, as a result of the danger of creating rhinitis medicamentosa. It ought to just be utilized for a short training course (less than 7-10 days) to minimize severe nasal clog, while co-administering other drugs such as nasal steroids [19].

Table 2. Components of a complete history and physical examination for suspected rhinitis [1].

History	Physical examination
Personal • Nasal itch • Rhinorrhea • Sneezing • Eye involvement • Seasonality • Triggers **Family** • Allergy • Asthma **Environmental** • Pollens • Animals • Flooring/upholstery • Mould • Humidity • Tobacco exposure **Medication/drug use** • Beta-blockers • ASA • NSAIDs • ACE inhibitors • Hormone therapy • Recreational cocaine use **Quality of life** • Rhinitis-specific questionnaire **Comorbidities** • Asthma • Mouth breathing • Snoring • Sinus involvement • Otitis media • Nasal polyps • Conjunctivitis **Response to previous medications** • Second-generation oral antihistamines • Intranasal corticosteroids	**Outward signs** • Mouth breathing • Rubbing the nose/transverse nasal crease • Frequent sniffling and/or throat clearing • Allergic shiners (dark circles under eyes) **Nose** • Mucosal swelling, bleeding • Pale, thin secretions • Polyps or other structural abnormalities **Ears** • Generally normal • Pneumatic otoscopy to assess for Eustachian tube dysfunction • Valsalva's maneuver to assess for fluid behind the ear drum **Sinuses** • Palpation of sinuses for signs of tenderness • Maxillary tooth sensitivity **Posterior oropharynx** • Postnasal drip • Lymphoid hyperplasia ("cobblestoning") • Tonsillar hypertrophy **Chest and skin** • Atopic disease • Wheezing

ASA: acetylsalicylic acid; NSAIDs: non-steroidal anti-inflammatory drugs; ACE: angiotensin-converting enzyme; OTC: over-the-counter

CONCLUSION:

AR is worth dealing with efficiently, even when it is part of a myriad of allergic conditions; as the nose is the gateway to the respiratory system tract, good rhinitis control can facilitate control of signs in other places. AR, if badly managed, results in bothersome signs and effect on daily activities, quality of life and on other areas of the respiratory tract, such as ears, sinuses, throat and lungs. Feasible reasons for difficult-to-treat instances consist of physician factors such as misdiagnosis and under treatment or patient aspects such as insufficiency of concordance with treatment. This can be decreased by information brochures on specific irritant evasion, the proper strategy in the application of nasal preparations and explanation of prospective adverse effects. Clearly written therapy strategies work, particularly if a number of preparations are suggested. Management of AR is a significant element of the practice for primary care centers. It is important that international standards have clear criteria for the medical diagnosis of AR and functional recommendations for reliable treatment. Local alterations might require to be made, however the requirement for diagnosis and efficient therapy of AR must not be compromised. Suitable patient education by doctors with a mutual

understanding of the nature of rhinitis and the available therapy alternatives will certainly make the most of patient conformity and improve treatment end results.

REFERENCES:

1. Small P, Frenkiel S, Becker A, Boisvert P, Bouchard J MD, Carr S, Cockcroft D, Denburg J, Desrosiers M, Gall R, Hamid Q, Hébert J, Javer A, Keith P, Kim H, Lavigne F, Lemièr C, Massoud E, Payton K, Schellenberg B, Sussman G, Tannenbaum D, Watson W, Witterick I, Wright E. The Canadian Rhinitis Working Group. Rhinitis: A practical and comprehensive approach to assessment and therapy. J Otolaryngol. 2007;36(Suppl 1):S5–S27.
2. Kim H, Kaplan A. Treatment and management of allergic rhinitis [feature] Clinical Focus. 2008. pp. 1–4.
3. Guerra S, Sherrill D, Martinez F, Barbee RA. Rhinitis as an independent risk factor for adult-onset asthma. J Allergy Clin Immunol. 2002;109:419–425. doi: 10.1067/mai.2002.121701.
4. Lee P, Mace S. An approach to allergic rhinitis. Allergy Rounds. 2009. p. 1.
5. Chaplin DD. 1. Overview of the human immune response. J Allergy Clin Immunol. 2006;117:S430–S435.
6. Broide DH. The pathophysiology of allergic rhinoconjunctivitis. Allergy Asthma Proc. 2007;28:398–403.
7. Prussin C, Metcalfe DD. 5. IgE, mast cells, basophils, and eosinophils. J Allergy Clin Immunol. 2006;117:S450–S456.
8. Kay AB. Allergy and allergic diseases. Second of two parts. N Engl J Med. 2001;344:109–113.
9. Togias A. Unique mechanistic features of allergic rhinitis. J Allergy Clin Immunol. 2000;105:S599–S604.
10. Gerth van Wijk RG, de Graaf-in't Veld C, Garrelds IM. Nasal hyperreactivity. Rhinology. 1999;37:50–55.
11. Linneberg A, Henrik Nielsen N, Frolund L, Madsen F, Dirksen A, Jorgensen T. The link between allergic rhinitis and allergic asthma: a prospective population-based study. The Copenhagen Allergy Study. Allergy. 2002;57:1048–1052.
12. Madonini E, Briatico-Vangosa G, Pappacoda A, Maccagni G, Cardani A, Saporiti F. Seasonal increase of bronchial reactivity in allergic rhinitis. J Allergy Clin Immunol. 1987;79:358–363.
13. Corren J, Adinoff AD, Irvin CG. Changes in bronchial responsiveness following nasal provocation with allergen. J Allergy Clin Immunol. 1992;89:611–618.
14. Togias A. Rhinitis and asthma: evidence for respiratory system integration. J Allergy Clin Immunol. 2003;111:1171–1183. quiz 1184.
15. Wang DY, Niti M, Smith JD, Yeoh KH, Ng TP. Rhinitis: do diagnostic criteria affect the prevalence and treatment. Allergy. 2002;57:150–154.
16. Ryan D, Grant-Casey J, Scadding G, Pereira S, Pinnock H, Sheikh A. Management of allergic rhinitis in UK primary care: baseline audit. Prim Care Respir J. 2005;14:204–209.
17. Crobach MJ, Hermans J, Kaptein AA, Ridderikhoff J, Petri H, Mulder JD. The diagnosis of allergic rhinitis: how to combine the medical history with the results of radioallergosorbent tests and skin prick tests. Scand J Prim Health Care. 1998;16:30–36.
18. Demoly P, Allaert FA, Lecasble M. ERASM, a pharmacoepidemiologic survey on management of intermittent allergic rhinitis in every day general medical practice in France. Allergy. 2002;57:546–554.
19. Brozek JL, Bousquet J, Baena-Cagnani CE, Bonini S, Canonica GW, Casale TB, van Wijk RG, Ohta K, Zuberbier T, Schünemann HJ. Allergic Rhinitis and its Impact on Asthma (ARIA) guidelines: 2010 revision. J Allergy Clin Immunol. 2010;126:466–476.
20. Van Hoecke H, Vastesaeger N, Dewulf L, Sys L, van Cauwenberge P. Classification and management of allergic rhinitis patients in general practice during pollen season. Allergy. 2006;61:705–711.
21. Scadding GK, Richards DH, Price MJ. Patient and physician perspectives on the impact and management of perennial and seasonal allergic rhinitis. Clin Otolaryngol Allied Sci. 2000;25:551–557.
22. Kasje WN, Timmer JW, Boendermaker PM, Haaijer-Ruskamp FM. Dutch GPs' perceptions: the influence of out-of-pocket costs on prescribing. Soc Sci Med. 2002;55:1571–1578.
23. Fokkens WJ. Who should treat patients with seasonal allergic rhinitis? Allergy. 2002;57:469–471.
24. Zhang L, Chew FT, Soh SY, Yi FC, Law SY, Goh DY, Lee BW. Prevalence and distribution of indoor allergens in Singapore. Clin Exp Allergy. 1997;27:876–885.
25. Wang DY, Chan A, Smith JD. Management of allergic rhinitis: a common part of practice in primary care clinics. Allergy. 2004;59:315–319.