

Allergic and non-Allergic rhinitis and nasal polyposis

أم د حيدر السرحان

Rhinitis is defined as at least two symptoms from a list comprising of rhinorrhoea, blockage, sneezing and nasal itching.

Rhinitis is divided into infectious and noninfectious and the latter further -subdivided into allergic (AR) and non-allergic rhinitis (NAR).

Allergic rhinitis

(AR) is an immunoglobulin E(IgE)-mediated, type 1 hypersensitivity reaction in the mucous membranes of the nasal airways to an allergen or allergens. It can be either seasonal (e.g., hay fever) or perennial (sometimes with seasonal exacerbations).

Some patients are atopic. Atopic means that they have a genetic susceptibility to form an immune (allergic) response to a range of commonly found allergens. An allergen that causes AR may also cause allergic asthma, atopic eczema (allergic dermatitis) and allergic conjunctivitis.

Pathophysiology:

Sensitization to an allergen occurs in genetically predisposed people who are, to a variable degree, atopic. Allergens are captured by dendritic allergen-presenting cells. This activates T-helper (Th) cells and these activated Th cells, called Th2 cells, release cytokines, the most important of which is interleukin 4 (IL-4). IL-4 drives B cells to produce allergen-specific IgE. This IgE binds through its high-affinity receptor, to effector cells such as mast cells and basophils. Subsequent contact of a nasal allergen with two molecules of allergen-specific IgE on the surface of a mast cell triggers the immediate release of inflammatory mediators such as histamine, tryptase and prostaglandin causing an immediate inflammatory response. Cytokines, most importantly IL-4 again, are also

released which permit the migration of leucocytes, particularly eosinophils, to the sensitised mucosa. These leucocytes in turn release their own cytokines leading to a late-phase inflammatory response.

Classification:

A. Intermittent. Symptoms are present fewer than 4 days a week or for fewer than 4 consecutive weeks.

B. Persistent. Symptoms present for 4 or more days a week or for 4 or more consecutive weeks.

Intermittent and persistent can each be subdivided according to severity to:

1. Mild. Symptoms are not troublesome and in particular do not cause sleep disturbance, impair daily leisure, do not impair sport activities and do not impair school or work.

2. Moderate to severe. Symptoms are troublesome and in particular disturb sleep, impair daily -activities and impair attendance/performance at school or work.

Clinical Features:

The patient suffers from rhinorrhoea, nasal irritation and sneezing, nasal obstruction and nasal congestion (nasal breathing is fine, but patients have midfacial pressure and feel blocked). The throat may be itchy or irritable and there may be associated itchy, red, watering eyes and urticarial upper and/or lower lid oedema that collectively can be mistaken for early periorbital cellulitis. This allergic conjunctivitis occurs in about 60% of those with AR and is the symptom that most reliably differentiates AR from other types of rhinitis.

On examination, the nasal mucosa usually appears moist, pale and swollen, and the -turbinates hypertrophied. Sometimes the mucosa is injected, and the turbinates may have a blue hue. Signs of chronic rhinosinusitis may be present with or without nasal polyps.

Investigations:

Skin tests The epidermal prick test and the intradermal injection test use an allergen placed on the skin of the flexor aspect of the forearm. If the patient has an allergy to this, then a wheal and flare will come up within 20 minutes.

Blood tests Total plasma IgE levels may be measured in the plasma radioimmunosorbent test (PRIST) and IgE to specific allergens in the radioallergosorbent test (RAST).

Nasal smears An increase in eosinophils in a nasal smear may occur in allergic rhinitis but is not specific.

Nasal challenge A drop of the suspected allergen squeezed into the nose may cause rhinitis symptoms. The obstructive effect can be measured objectively by rhinomanometry pre- and post-challenge.

Management:

Avoidance or reduced exposure to the precipitating allergen is perhaps the most important line in the treatment.

Selective oral antihistamines selectively block histamine receptors and cause minimal or no drowsiness and can be given once daily (e.g., cetirizine, fexofenadine and loratadine).

Topical steroid sprays and drops are now considered to be the cornerstone in the treatment of rhinitis. They are safe and effective for nasal blockage, nasal itchiness, watery rhinorrhea and sneezing. Crusting and bleeding are the main side effects. Systemic absorption is less than 2% for the prescribable nasal steroids and they are therefore safe to use long term in most patients. Examples include fluticasone, mometasone sprays.

Oral steroids for 7 to 10 days are very effective in rapidly reducing symptoms and, by reducing mucosal oedema, they allow nasal sprays to be more widely distributed within the nasal -cavity. They may be used in poor responders to nasal steroids/antihistamine sprays.

Sodium cromoglycate stabilises mast cell membranes and therefore prevents the release of the allergic response mediators.

Desensitisation immunotherapy involves exposure to regular small but increasing doses of an allergen to produce tolerance to the allergen by producing blocking IgG antibodies.

Leukotriene receptor antagonists appear to have an additive effect to intranasal corticosteroids in controlling seasonal AR, particularly in patients with asthma.

Surgical treatment in form of inferior turbinate sub-mucosal diathermy and more recently inferior turbinate laser turbinoplasty.

Non-Allergic Rhinitis

Psychological and emotional

Endocrine (puberty, menstruation and pregnancy).

Drugs (angiotensin-converting enzyme [ACE] inhibitors, β -blockers, methyldopa, aspirin and oral contraceptives).

Climate changes in humidity and temperature.

Alcohol induced

Smoking (tobacco nasal mucosal inflammation)

Clinical Features:

Anterior and posterior rhinorrhea: those patients usually complaining from excessive post nasal drip , nasal discharge anteriorly is also common

Nasal obstruction: complain of marked blockage when lying flat trying to sleep.

Investigations:

diagnosis of exclusion, and the aim of investigations is to identify other causes of rhinitis. Immunoglobulin E (IgE) estimation by PRIST and RAST and skin testing can be used to indicate allergy. If a systemic inflammatory process is suspected, an anti-neutrophil cytoplasmic antibody (ANCA) and ACE assay may be indicated.

Treatment:

A short course of oral steroids may be indicated when there is marked nasal mucosal oedema and obstruction

Nasal steroid sprays also used Topical ipratropium bromide has an anticholinergic effect and may reduce watery rhinorrhea.

Systemic sympathomimetics can be helpful (e.g. pseudoephedrine), though they may produce unpleasant side effects such as dry mouth, constipation and excitability. They should not be used long term or in children.

Surgical treatment by Vidian neurectomy This operation was proposed for the relief of watery rhinorrhea.

Nasal Polyps:

a marked prolapsing and swelling of the sinus and nasal mucosa that herniate in the nasal cavity

Clinical Features:

Nasal polyps may be asymptomatic and found incidentally but, even when they are small, patients may complain of a feeling of pressure high in the nose and may have hyponasal speech. As the polyps enlarge there is increased nasal obstruction and usually anterior or posterior rhinorrhoea. Airflow over the olfactory cleft and sphenoethmoidal recess is increasingly impeded causing hyposmia and often anosmia with concomitant hypoageusia. Headaches, pressure sensation in the face and secondary infective sinusitis may occur. In severe cases the polyps may

be visible at the external nares and widening of the intercanthal distance may occur (telecanthus).

Investigations:

CT scan is needed prior to Endoscopic sinus surgery

Investigations to rule out AR

Treatment:

Saline irrigation will help to thin and wash away the inspissated mucous that patients produce, allowing nasal steroids to then coat nasal polyps more reliably. Steroid drops, an increased dose of nasal steroid spray or a short course of oral steroids is necessary, A long (at least 12 weeks) course of doxycycline may reduce polyp size and sensation of blockage as well as reducing post nasal drip.

surgical treatment: endoscopic polypectomy under either local or general anaesthetic. A more radical clearance by means of an endoscopic fronto-ethmoidectomy leads to better longer-term disease control.

post-operative Long-term saline nasal douching is recommended. Intranasal steroids reduce recurrence in the first year and may make a difference to the recurrence rate long term .