Allergic Rhinitis (Hay fever)

Introduction

- Characterised by inflammatory changes in the nasal mucosa caused by exposure to an inhaled allergen to which an individual has become sensitised (type I hypersensitivity reaction)
- Sensitisation phase – allergen exposure and B cells produce allergen-specific IgE
- Early phase – subsequent exposure to allergen results in IgE cross-linking and mast cell degranulation
- Late phase – inflammatory cells infiltrate mucosa with persistent symptoms = **OBSTRUCTION**
- AR forms part of unified allergic airway – allergic asthma / conjunctivitis
- Allergen = antigen (usually protein) that causes allergic diseases
- Hypersensitivity = altered immune response to an antigen that can cause damage
- Atopy = tendency to become sensitised and produce antigen specific AB's (IgE) in response to an ordinary exposure to allergens
- Not all sensitised people are symptomatic
- An Atopic person (40% in the developed world):
    - Usually have high serum total IgE
    - Strong familial tendency
    - Increased susceptibility to allergic diseases (AR / Atopic dermatitis)

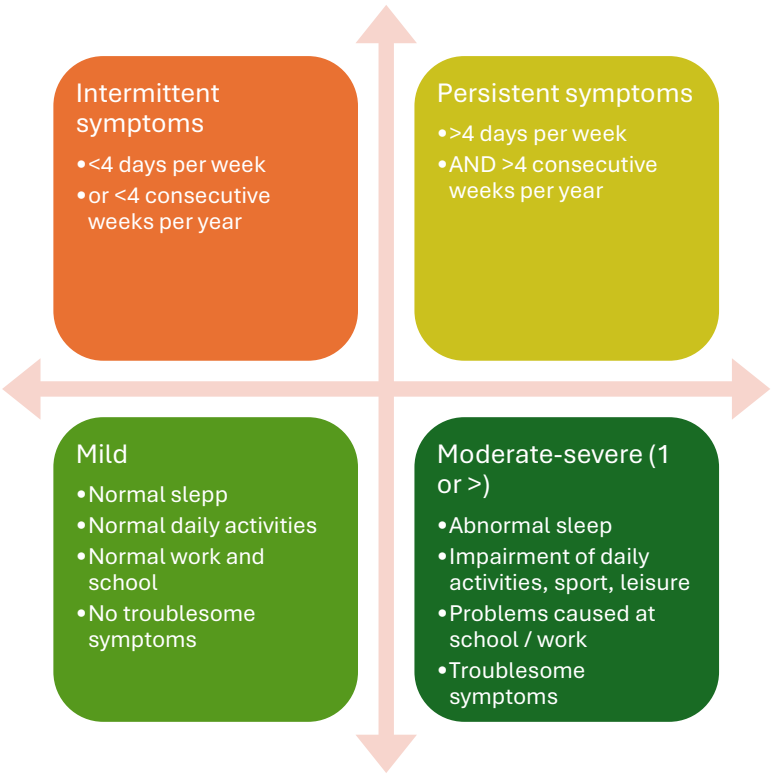
Development of sensitisation

- Mast cells and Basophils are the primary effector cells
- IgE related immediate allergy is associated with early and late responses
- Early phase symptoms are due to preformed mediators
- Late phase due to newly formed mediators and infiltration of other leucocytes
- Involves Th2 cytokines that activate eosinophils and other leukocytes
- Chronic allergic inflammation is associated with tissue destruction and remodelling
- T cells are the only cells capable of recognising an antigen presented by an antigen presenting cell (APC) thus central in the development of allergic disease
- An allergen is presented to the Th2 cell by the Dendritic cell à release of inflammatory mediators with mast cell degranulation and Histamine release (amongst other mediators)

Immune response:

- Initially naïve T helper cells (CD3+/CD4+) may mature in different ways depending on the environment
- TH1 response
    - Th1 cells differentiate in the presence of IFN-ɣ and IL-12 à they produce IFN-ɣ and IL-2
    - High dose antigen exposure as with injectable, favours a Th1 response (how desensitisation works)
- TH2 response
    - Th2 cells differentiate in the presence of IL-4 and produce IL-4 / IL-5 / IL-13
    - Recruitment and activation of Th2 cells with above cytokines is typical in AR / Asthma
    - Low dose antigen exposure via mucosal surface with an inhaled antigen is typical of a Th2 response

Classification



History

Atopic conditions (Pt or family - if parents are atopic then 3-6 x increased risk)

- Childhood eczema / Asthma
- Atopic march = infantile eczema and AR and Asthma
- Intermittent vs Persistent symptoms

Identifiable allergen (house dust mite / pollen / grass /fungus / dander)

- Difficult to modify exposure

Clinical diagnosis with 2 or more of the following symptoms:

- Anterior or posterior rhinorrhoea (watery)
- Sneezing
- Nasal obstruction
- Itching
    - Other symptoms - itchy eyes, pharyngeal itch, reduced smell, cough and sore throat

Any prior treatment and response to it?

Red flags - **NOT** typical of AR and should prompt further investigation

- Unilateral symptoms
- Purulent discharge
- Epistaxis
- Pain

Examination

- Allergic shiners / Dennie-Morgan lines

- Allergic salute
- Enlarged inferior turbinates
- Inflamed mucosa over IT's (dull/blue/purple)

Investigation

Can be correlated with diagnostic tests if allergen not clear from history

- Skin prick tests
- Specific IgE levels in blood
    - Can do individual allergen of bundle tests (phadiotop for inhalent / FX5 for foods)

Both have similar sensitivity and specificity

Management

- Allergen avoidance if possible
    - Animal dander / pollen / grasses / fungus / house dust mite
    - Stop smoking / exposure to 2nd hand smoking
- Symptomatic therapy (pharmacotherapy)
- Desensitisation

Medical treatment

- Systemic +- topical Antihistamine – act on H1 receptors
    - Effective in partial suppression of immediate allergic response
    - Little to no effect in the late response

- Topical Corticosteroids (nasal spray) - **Cornerstone of treatment**
    - Act on intracellular receptors causing anti-inflammatory effect
    - Allergen-induced Th2 proliferation and cytokine release is very sensitive to low concentrations of corticosteroid
    - Highly effective on late response (Th2 related)
    - No effect on the immediate response
    - Do not confuse it with other nasal sprays
        - Iliadin, Otrovin, Drixine, Sterimar, Salex
    - You need to use it everyday
        - Prophylactic treatment
    - 6% incidence of nasal bleeding
        - Technique of spray
        - Other methods of delivery (Ampules / Nasules)

Educate on technique of use of corticosteroid spray

Shake bottle and prime bottle. Bend head forward and direction of spray (left hand for right nose, right hand for left nose and aim for eye on the same side)

As you spray inhale gently and exhale through your mouth

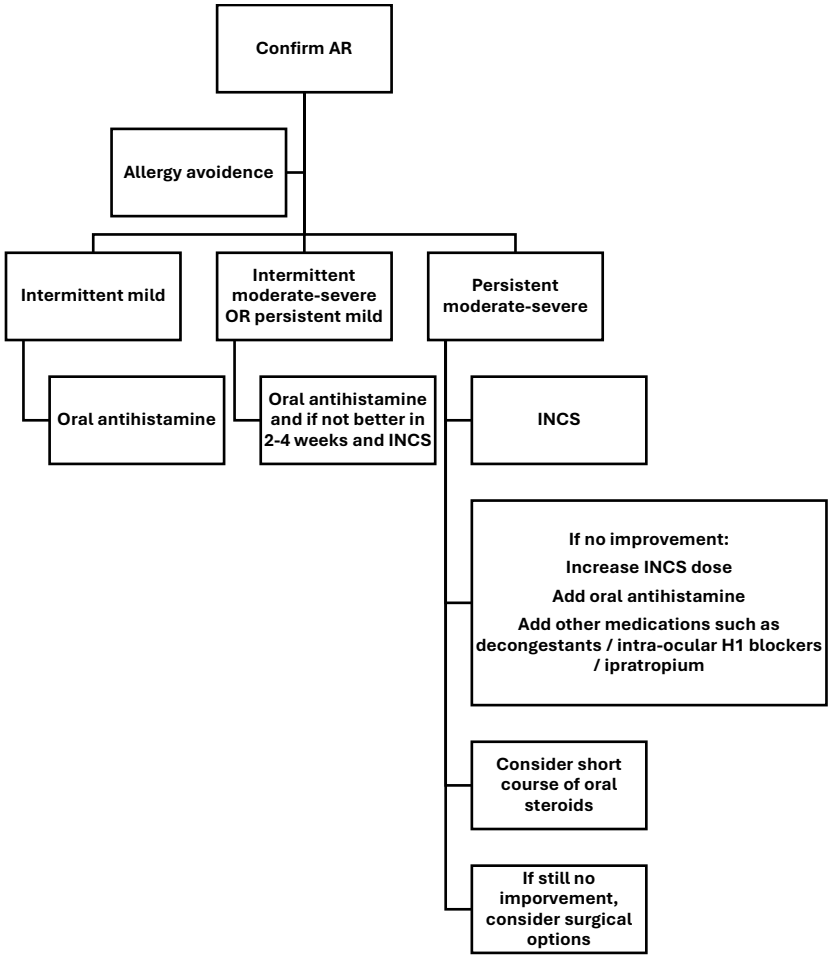
Allergen-specific immunotherapy (Desensitisation)

- Only curative therapy option and highly effective (Cochrane review*)
- Involves giving increasing doses of an allergen to suppress the symptoms when re-exposed to that allergen
- Risk of anaphylaxis thus perform in appropriate setting
- 2 major routes of administration
    - Sublingual immunotherapy (SLIT)

- Subcutaneous immunotherapy (SCIT)
- Mechanisms of action
    - Desensitisation of mast cells and basophils
    - Upregulation of blocking antibodies that bind to allergen (reduce antigen presentation)
    - More regulatory T and B cells (Fewer tissue mast cells/eosinophils)

Other treatment

- Sodium cromoglycate as eye drops (mast cell stabiliser) – useful for allergic ocular symptoms
- Topical anticholinergic (ipratropium bromide) – if rhinorrhoea still troublesome with INCS
- Leukotriene receptor antagonist (montelukast) if co-morbid Asthma (especially in children)
- Subcutanous anti-IgE antibodies (Omalizumab)may be effective in severe disease
    - Cost limits its use (R14 000 per/month and up)



Conclusion

- Antihistamine – mild/intermittent disease
- INCS is mainstay
- If rhinorrhoea still troublesome – topical anticholinergic (ipratropium bromide)
- Rarely oral corticosteroid short-course (e.g. 20mg prednisone Dly x 5/7)
- Immunotherapy / desensitisation (Treat the cause)
    - Sublingual / subcutaneous
- Consider ENT referral if poor / inadequate response to medication