

NOSE II

Objectives:

- > Acute & chronic rhinitis.
- ➤ Allergic & non-allergic rhinitis.
- > Vestibular & furunculosis .
- ➤ Nasal polyps (allergic & antrochoanal) etc.
- ➤ Radiology illustration (e.g. CT scan)

[Color index : Important | Notes | Extra]

Resources: Slides+433team+Notes

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((The doctor's lecture was only about nasal polyps but the lecture objectives include things other than the nasal polyps covered by 432 team.))

Rhinitis:

- Rhinitis refers to inflammatory changes in the nasal mucosa. As the nasal mucosa is continuous over the nose and sinuses, there is nearly always some inflammatory change in the sinuses as well. Hence Rhinosinusitis is a better term.
- Continuing inflammatory change or repeated episodes of recurrent rhinosinusitis so that episodes merge one with the other are common Chronic Rhinosinusitis (CR).
- Continuing mucosal inflammation causes polypoid swelling of the nasal lining (Nasal Polyps, hence the close association between Chronic Rhinosinusitis and Nasal Polyps "CRNP").

Acute Rhinitis:

- The **common cold** is the result of viral infection but secondary bacterial infection may supervene.
- It is self-limiting and no treatment is required other than an antipyretic, such as paracetamol. Discourage the prolonged use (more than 5 days) of vasoconstrictor nose drops owing to their harmful effect on the nasal mucosa (rhinitis medicamentosa).
- Many patients use menthol inhalations, systemic decongestants and a variety of cough linctus preparations, and find these helpful in controlling symptoms, but evidence of any sustained benefit is weak.
- (common cold & influenza & Para influenza)

→ Etiology:

- **Virus:** (rhinovirus, coronavirus, adenovirus, Para influenza virus, respiratory syncytial virus, enterovirus).
- Nasal obstruction: Pyrexia.
- Bacterial: Streptococcus pneumoniae, Streptococcus pyogenes, Haemophilus influenzae and Moraxella catarrhalis.

→ Clinical stages:

- Dry prodromal stage (few hours).
- The Catarrhal stage (few days).
- Mucous stage (3-5 days).
- Resolution stage (5 days- 1 week).
- Secondary bacterial infection.

→ Treatment:

- o Prophylactic: avoid contact with patient
- Therapeutic:
 - Rest
 - Analgesics
 - Decongestant
 - Antibiotic
 - We might have pus in sever viral infection, it's dead
 WBC, but antibiotic is rarely given for viral infection.
 - *Acute rhinitis is < 3 weeks, chronic is >3 months*

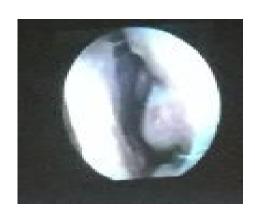


Chronic Rhinitis:

Classification:

> Specific:

- Syphilis.
- Wegner's granuloma.
- Medline lethal granuloma.
- Sarcoidosis.
- Mycobacteria (TB rare).



➤ Non-specific:

- Atrophic rhinitis:
 - **Definition:** chronic inflammation of the nasal mucosa with atrophy of various nasal constituents.
 - Etiology:
 - Not fully known
 - Infection
 - Endocrine or vitamin disturbance
 - Types:
 - **Primary** (without any interference).
 - Secondary (usually related to surgery).

Traid of atrophic rhinitis:

- Fetor (bad smell)
- Crusting
- Atrophy of the nasal structures involving the bone as well, mucosa will be fragile and thinner.
- *If you have this traid, you must take swab and send for culture

Primary atrophic rhinitis	Secondary atrophic rhinitis	
 Clinical feature: Foul stench (very bad smell but patient may not notice due to adaption of smelling sensation) Epistaxis (due to atrophy and crusting) Sensation of obstruction (big nasal cavity without turbulence = feeling of obstruction called roomy nasal syndrome or empty nasal syndrome) Pathology: 1.degenration of epithelium glands Thick crust in the nose-infected Foul smelling 2-atrophy of the bony turbinates 	 Secondary to sever deviated septum, syphilis, lupus, excessive operation (turbinectomy, which is less common nowadays). Main goal of medical treatment: restoration of nasal hydration and minimize crusting. 	
 Treatment: Removal of the crust Glucose 25% in glycerin drops to lubricate the nose Local or systemic antibiotic if bacterial infection Surgical measures if severe and lead to obstruction 		

- → Anti-evaporation compounds (not given in a big quantity to avoid going to the lung and cause pneumonitis or it may emboli):
 - Glycerin
 - Mineral oil
 - Methanol mixed with paraffin
 - Odor masking agents, such as rose oil or methanol

→ Systemic or oral therapies:

- Aminoglycoside for Pseudomonas, a strictly aerobic, gram-negative rod of relatively low virulence.
- Tetracycline
- Fluoroquinolones

→ Surgical therapies:

- Degenerative operations
- Volume reduction operations
- Nasal closure operations (close then open).

Simple chronic rhinitis:

Etiology:

- Neighboring infections e.g. chronic tonsillitis. "GERD can cause it as well"
- Adenoids "obstructing normal mucosal secretions => stagnation and good environment for bacteria to grow.
- Vasomotor rhinitis "happen as a result of autonomic disturbance".
- Chronic irritation e.g. dust, smoker.
- Swelling of inferior turbinate.
- **Treatment:** Correction of any predisposing factors.

Hypertrophic rhinitis:

 Etiology: Permanent hypertrophic changes due to advanced stage of simple chronic rhinitis.

Rhinitis medicamentosa:

- Etiology: Induced by sympathomimetic nasal decongestant drops" like atropine"
- If we need to use sympathomimetic nasal decongestant drops then it must be not more than 5days".
 - ✓ Q: What if you need to use decongestant for more than 5days?
 - ✓ Answer: We have to give systemic sympathomimetic decongestants like pseudoephedrine "No rebound effect".
- Clinical features: Like simple chronic rhinitis.
- Treatment: Like simple chronic rhinitis + Reduction of inferior turbinate.

Allergic Rhinitis:

- All rhinitis has allergic factors except 20-30% are not and called non-allergic rhinitis or vasomotor rhinitis.
- Due to inhalation of the allergen.
- The disease is hereditary AR Antigens are usually wind-borne [e.g.; grass & tree pollens], HDM (house dust mite), fungi, dog &cat dander.

Pathophysiology:

- The nasal mucosa is at the entrance to the respiratory tract. It is made up of ciliated epithelium and produces a mucus blanket which helps protect the airway from inspired pollutants, allergens and infective agents.
- The epithelium is continuous with that of the rest of the respiratory system and is subject to much the same pathologies.
- The allergen induces production of IgE antibodies which on subsequent exposure bind with the allergen to form antigen—antibody complexes. These complexes then attach to mast cells in the nasal epithelium, causing the cells to rupture and release inflammatory mediators including histamine (Type 1 allergic response).
- An intense local inflammatory reaction ensues with oedema and secretion of mucus. The
 nose may now become sensitive to irritants in inspired air, so that the slightest stimulus
 will cause symptoms to recur.
- Allergic rhinitis primarily occurs in atopic individuals.
- Atopy is a general increased sensitivity to the production of Immunoglobulin E in response to small amounts of allergens-typically specific proteins.
- Atopic people may develop both rhinitis and asthma ('sneezles and wheezles') and they
 often have a strong family history of these disorders and of eczema.

Clinical features:

- Diagnosis is clinical, You don't usually need X-rays or scans. The main symptoms are:
 - A feeling of nasal congestion.
 - Nasal airflow obstruction.
 - Rhinorrhoea or a watery nasal discharge.
 - o Sneezing.
 - Reduced or absent sense of smell (hyposmia or anosmia).

***** Types:

> Seasonal Allergic rhinitis:

• Start Early Spring by [tree pollen] then in Midsummer by [grass pollen] and end in autumn by [molds].

> Perennial Allergic rhinitis:

- Causes:
 - The house dust mite is the most common cause.
 - A mountain hut in Sweden. (Go there to escape AR!)
 - Fungi, animal hair, house dust& mites.
 - Houseplants: [primulas & rose].
 - Food: [fish, strawberries, nuts, eggs, milk, &flour].

➤ Occupational allergies:

• [Bakers, hairdressers &painters] (e.g.; latex, powders, paint vapors).

-The classification "seasonal" and "perennial" allergic rhinitis has been changed to "intermittent" and "persistent" allergic rhinitis

-Seasonal allergic rhinitis occurs only when pollens in the air (hay fever).

-Perennial rhinitis occurs all year round & can be a myriad of substances, although house dust mite is high on the list

-Nonorganic substances can induce inflammatory reaction and nonspecific irritants like: smoke and dust >> Vasoactive substances (the reaction is not IgE mediated but it's IgG).

-Physical factors can affect the mast cell like: Temperature change& alcohol.

→ The Classic Reaction:

- Type1 IgE mediated reaction, produced in Plasma cells and regulated in T-Lymphocytes.
- IgE has a crystallized fraction that bind to MC (mast cell) then release protein (Fab).
- Fab +Antigen lead to triggering of mast cell degranulation and release of material like (Histamine, Leukotriene, Prostaglandin).
- These substances cause: Mucosal edema &nasal secretion.

Diagnosis of AR:

- Detailed medical history.
- ENT examination.
- Other tests as appropriate: allergy tests, endoscopy, nasal smear, nasal swab, radiology, nasal airway assessment, olfaction, and blood tests.

→ AR Symptoms:

- "SNEEZERS AND RUNNERS": Itchy nose, sneezing, watery rhinorrhea, nasal congestion (variable), diurnal rhythm (worse during day), often associated conjunctivitis.
- "BLOCKERS": Little or no sneezing, thick catarrh (with post nasal drip), no itch, and constant symptoms - possibly worse at night.

→ Examination:

Allergic crease.

• Nasal mucosa: livid & pale.

• The turbinates: swollen.

• Clear secretion: +++.







→ Investigation:

Skin prick testing	Nasal Cytology	 Intracutaneous tests
RAST (radioallergosorbent test)	•IgE	 Mucociliary clearance
*Typical history	Rhinomanometry	 Rhinoscopy tests
Patch tests	Nasal provocation test	P.N.S. CT

• *PNS CT = CT of the Para nasal sinuses

→ DDX of AR:

- Vasomotor rhinitis .
- Common cold.
- Cerebrospinal rhinorrhea.
- Polyps.
- Granulomas e.g. Wegener's Granulomatosis.
- Mechanical factors: deviated septum, hypertrophic turbinates, and foreign bodies.
- Coryza.
- Tumors.

→ Treatment:

Causal Treatment	Symptomatic Treatment (imp)
 Immunotherapy or Desensitization. 	Antihistamines.
Allergen Avoidance.	 Topical Steroids & Cromoglycate.
·Local or systemic inhibitions of H substance.	Systemic Steroids.
(4) And dependence of the second of the control	Nasal Decongestant.
	Anticholinergic.
	•Antibiotics. (after culture & sensitivity)
	•Polypectomy & turbinoplasty.

Rhinitis sicca anterior:

Pathogenesis:

➤ Anterior part of the septum usually exposed to hot, dry weather. So trauma or dryness >> irritation to this part >> Crusts formation and attempt to remove it >> Nasal bleeding>> septal perforation.

• On examination:

- Nasal septum is dry
- o Mucosal surface is: Raw, roughened, & granular.
- Crustation >>ulceration>> Septal perforation

Differential Diagnosis:

- Chemical injury (Chromium workers)
- o latrogenic septal perforation
- o Trauma
- Lupus
- Leprosy
- o syphilis

• Treatment:

- Nasal ointments
- Septal perforation closure

• Prognosis:

- Good prognosis
- Regresses with time
- Sometimes → bronchial asthma

• Complications:

- o L.R.T.
- Nasal sinus
- Nasal or sinuses polyps

• Indications for surgery:

- Anatomical abnormalities
- Excessive mucosal swelling
- Presence of irreversibly diseased tissue

Surgical procedure:

- Polyp removal
- Removal of adenoi
- Correction of septal deformities
- Turbinate surgery
- Sinus drainage

Pregnancy Rhinitis

- Nasal swelling & obstruction
- Start at the 2nd half of pregnancy
- Resolve after delivery

Rhinitis Medicamentosa

- Reversible or irreversible damaged mucosa caused by topically or systemically applied drugs:
 - Hyperplastic Rhinitis
 - o Dryness of the nasal mucosa
 - Toxic Rhinopathy (Vasoactive substances)

Atrophic Rhinitis & ozena

Atrophic rhinitis foul smell = Ozena

> Types:

- o Primary: rare
- Secondary: common, and it's due to massive cutting of the turbinate in surgery, trauma or Occupational exposure to: Glass, wood, asbestos, etc.
- Mainly in women, at puberty, Flattened nose & broad Face (due to fullness of crustation)

Examination:

- o Greenish-yellow or brownish-black crusts.
- Wide nasal cavity.
- Atrophic mucosa & dry: Sub epithelial layer fibrosis.
- Fetid secretion &crusts (Ozena.)
- Anosmia & social problem.
- Nasal obstruction.

[&]quot;Acute intoxication in infants & small children"

Pathogenesis:

- Unknown cause but is multifactorial
- Common in Orientals than in whites than in blacks
- Respiratory epith. >> sq. metaplasia
- Destroyed mucociliary cleaning system
- Bacterial proteolysis decomposed the thick & gluey secretions

★ Differential Diagnosis:

- Atrophic rhinitis with fetor (ozena)
- Tumors of the Nose & Sinuses
- Purulent Rhinitis & Sinusitis
- Nasal diphtheria & Nasal Tuberculosis
- Gumma due to stage III Syphilis
- Rhinolith & foreign body
- glanders

♦ Treatment:

Conservative(better than surgery)	Operative
Nasal douching.	Bolstering of the Nasal Mucosa(Cartilage or Bone chips).
Alkaline nasal lotion.	Median Displacement of the lateral nasal wall by internal
Greasy ointments.	rotation of the mobilized lateral nasal wall.
Oily nasal drops, emulsions, or	
ointments.	
Steam inhalations.	
Osmotic Powders : Dextrose.	

Vestibulitis and furunculosis

Furunculosis of nasal vestibulitis:

Acute staphylococcal infection of hair follicle

- Vestibule: is lined by squamous cell epithelium not respiratory epithelium, so it can be affected by skin diseases like dermatitis and furunculosis.
- There will be pain, swelling, infection and sometimes fever.

Furunculosis: the same as vestibulitis, with pus.

Vestibulitis: more broad term than furunculosis, infection of skin and hair follicles.

Cellulites: skin infection without pus.

Clinical features:

Pain-tenderness-indurated swelling in the vestibule

Treatment:

Systemic antibiotics or topical depends if the patient is immune-compromised or extreme of age

Rx depend on immunity & symptoms & age (extreme age) If mild symptoms might give topical, if severe might give systemic in top of topical.

Complication:

Because head and neck have a valveless venous system, so it can go ante grade or retrograde, so it may push infection.

Cavernous sinus thrombosis (theoretical risk, called dangerous area), cellulitis of upper lip.



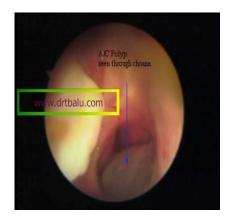
Nasal Vetibulitis

Nasal polyps

dr said nasal polyps are just a description not diagnosis

- Defined as: simple oedematous hypertrophic nasal mucosa. Can be unilateral / bilateral.
- Classification:
 - Simple nasal polyp
 - Fungal polyp
 - Malignant polyp
- Simple nasal polyp 'Also known as inflammatory polyp:'
 - Ethmoidal polyp
 - Antrochoanal polyp
- Antrochoanal polyp (present from the nose going to the nasopharynx).
- In general the ethmoidal and antrochoanal polyp are almost the same and have the same management.

Ethmoidal polyp	Antrochoanal polyp
Seen in adults	Seen in children and adolescents
Allergy is the common cause	Infection is the common cause
Multiple (bunch of grapes)	Unilateral
	Arises from maxillary antrum
Seen easily on anterior rhinoscopy	Seen commonly in post nasal exam
X ray PNS may show hazy ethmoids and normal maxillary sinuses	X ray PNS shows hazy maxillary antrum
Mostly bilateral	Usually unilateral
Recurrence is common	Recurrence is uncommon
Polypectomy	Caldwel luc surgery in recurrent cases



Fungal polyp

we divide the fungal polyp into invasive and noninvasive

• 5 Different types:

- Acute fulminant invasive
- Chronic invasive invasive
- Granulomatous invasive invasive
- Fungal ball non invasive
- Allergic fungal rhinosinusitis (AFRS) non invasive

Acute fulminant:

 Sudden onset and usually present in <u>immunocompromised</u> patients leading to serious consequences.

Chronic Invasive:

• Invades the basement membrane and go to the adjacent structures causing necrosis and ischemia.

Fungal Ball:

- Immunocompetent
- Fungal ball is tightly packed hyphae of aspergillus (common).
- Antifungal trt is not necessary

Allergic fungal rhinosinusitis (AFRS):

also <u>Immunocompetent</u> and its IGE mediated AFRS most commonly seen in Saudi Arabia. Bent's criteria

- Type I hypersensitivity (demonstrable)
- Nasal polyposis
- Heterodense mass lesion seen in CT scans
- Presence of eosinophilic mucin mixed with non invasive fungus
- + Fungal stain / culture

Clinical features:

- Nasal obstruction Unilateral / bilateral.
- Anosmia.
- Loss of taste.
- Rhinorrhoea watery / mucoid / mucopurulent.
- Headache.
- Broadening of nose (Frog face).





Examination:

- Smooth glossy grape-like multiple mass seen in anterior rhinoscopy.
- Insensitive on probing. Probe can be passed around the polyp (when touched the patient will not feel).
- Soft and mobile
- Polyp can be seen at the level of choana.
- Antrochoanal polyp can be seen exiting out of accessory ostium.

(possible **SAQ**)

What do you see in the photo?

-nasal polyp.

Give ddx?

- -Simple nasal polyp.
- -Allergic nasal polyp.
- Allergic fungal rhinosinusitis.
- -Malignancy
- -Inverted papilloma.



Posterior rhinoscopy

Differential diagnosis:

- Meningocele.
- o Angiofibroma.
- Sq cell carcinoma (squamous cell carcinoma is the most common malignancy in the nose).
- Enlarged turbinates.
- o Inverted papilloma (benign tumor).

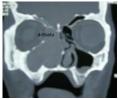
Radiology:

- Heterogenecity, remodelling > Allergic fungal.
- <u>No</u> heterogenecity, expanding, unilateral > <u>Malignancy</u>.









★ Medical Management:

- Steroids
- Normal saline irrigation
- Antibiotics (if acute)

In general start with medical treatment, if failed > surgical.
FESS risks (complications):

- 1. Bleeding
- 2. Synechiae formation
- 3. Orbital injury
- 4. Blindness
- 5. CSF leak

- 6. Nasolacrimal duct injury/epiphora
- 7. Diplopia
- 8. Orbital hematoma
- 9. Direct brain injury

• Surgery:

- Functional endoscopic sinus surgery (FESS)
- Endoscopic polypectomy
- o Caldwel Luc procedure

430 team:

Nasal polyps:

- Benign pedicle or sessile pale gray sacks of mucosa.
- Usually ethmoid sinus is affected.
- Etiology: infection, allergic, 90% eosinophil.
- Any age is possible but uncommon in children (exclude encephalocele).
- Male affected more than female but if the pt. is having asthma the ratio is equal.
- Mostly bilateral (if unilateral think of inverted papilloma, encephaolce or carcinoma).
- 100 to 1000 times histamine / serum (histamine is 100 times more than serum indicating mast cell degranulation in sinuses). "Causes of mast cell degranulation extreme temp. drugs, &complement factors."
- If the eosinophil >90% it suggests association with AR.
- 50% nasal polyps will develop asthma (proper treatment of the polyp will improve asthma).
- 80% have ASA or Sempter's triad (polyp, aspirin sensitivity and asthma).
- Nasal polyposis is chronic & recurrent disease.
- Patient have Miserable time and they respond well to Salicylate-free diet.

Clinical Feature:	examination:	Investigation
Nasal obstruction, decrease sense of smell, sneezing, rhinorrhea and Postnasal drip: clear, yellow or green	Bilateral pale, glistening gray sacks of polyp hanging from the sinuses.	CT scan (because FESS is usually indicated) Skin tests [H. dust or pollen]
(depend on degree of eosinophilia or infection). Hyponasal voice [permanent cold]. No pain unless secondary infection. No bleeding no discharge but if there is bleeding or serosanguinous discharge think of carcinoma	• Frog face (in a massive polyp) • differentiate between polyp and turbinate by pushing it with a probe if it's movable and painless >> polyp if not >> turbinate	In Child do: CT scanning to exclude (Encephalocele) Sweat test to exclude (Cystic Fibrosis)

Medical treatment **Surgical Treatment** Topical steroids: Nasal polypectomy(Partial or total ethmoidectomy) good in 50% First line of management Postoperatively Topical steroids :--Minimal time is 3 months 1- month course & review Head down position [drops] Aqueous spray :daily basis Patient must be aware of unwanted effects of steroid sprays Systemic steroids :Short reducing dose (it may lead to Avascular necrosis of the head of femur)













Antrochoanal polyp:

- Long pedicle unilateral solitary benign polypoidal lesions
- Arises in the intramural maxillary sinus cysts in the post. Aspect of the sinus
- Unknown etiology
- Can recur after treatment.
- Dumbbell shape
- Unilateral nasal blockage

Surgical treatment:

- Endoscopic nasal removal (by FESS)
- Caldwell-Luc procedure
- Snare Simple polypectomy: 20%recurrence

DDx:

Inf. Turbinate enlargement, polypoid rhinosinusitis, juvenile N angiofibroma, mucus R cyst, mucocele, benign or malignant nasopharyngeal tumors Preop. Components identification by its characteristic radiographic appearance.

Summary (from Toronto notes)

*Definition: inflammation of the lining (mucosa) of the nasal cavity

Table 10. Classification of Rhinitis

Inflammatory	Non-Inflammatory
Perennial non-allergic Asthma, ASA sensitivity Allergic Seasonal Perennial Atrophic Primary: Klebsiella ozena (especially in elderly) Acquired: post-surgery if too much mucosa or turbinate has been resected Infectious Viral: e.g. rhinovirus, influenza, parainfluenza, etc. Bacterial: e.g. S. aureus Fungal Granulomatous: TB, syphilis, leprosy Non-infectious Sarcoidosis Granulomatosis with polyangiitis Irritant Dust Chemicals Pollution	Rhinitis medicamentosa Topical decongestants Hormonal Pregnancy Estrogens Thyroid Idiopathic vasomotor

Table 11. Nasal Discharge: Character and Associated Conditions

Character	Associated Conditions	
Watery/mucoid	Allergic, viral, vasomotor, CSF leak (halo sign)	
Mucopurulent	Bacterial, foreign body	
Serosanguinous	Neoplasia	
Bloody	Trauma, neoplasia, bleeding disorder, hypertension/vascular disease	

Rhinitis medicamentosa: rebound congestion due to the overuse of intranasal vasoconstrictors. For prevention, use of these medications for only 5-7 d is recommended.

Congestion reduces nasal airflow and allows the nose to repair itself (i.e. washes away the irritants). Treatment should focus on the initial insult rather than target this defense mechanism.

Vasomotor Rhinitis

- Neurovascular disorder of nasal parasympathetic system (vidian nerve) affecting mucosal blood vessels
- Nonspecific reflex hypersensitivity of nasal mucosa
- Caused by:
 - > -Temperature change
 - > -Alcohol, dust, smoke
 - -Stress, anxiety, neurosis
 - > -Endocrine: hypothyroidism, pregnancy, menopause
 - > -Parasympathomimetic drugs
 - Beware of rhinitis medicamentosa: reactive vasodilation due to prolonged use (>5 d) of nasal drops and sprays (Dristan®, Otrivin®)

Clinical Features:

- Chronic intermittent nasal obstruction, varies from side to side
- Rhinorrhea: thin, watery
- Mucosa and turbinates: swollen
- Nasal allergy must be ruled out

Treatment:

- Elimination of irritant factors
- Parasympathetic blocker (Atrovent® nasal spray)
- Steroids (e.g. beclomethasone, fluticasone)
- Surgery (often of limited lasting benefit): electrocautery, cryosurgery, laser treatment or removal of inferior or middle turbinates
- Vidian neurectomy (rarely done)
- Symptomatic relief with exercise (increased sympathetic tone)

Allergic Rhinitis (Hay Fever)

Definition:

- Rhinitis characterized by an IgE-mediated hypersensitivity to foreign allergens
- Acute-and-seasonal or chronic-and-perennial
- Perennial allergic rhinitis often confused with recurrent colds

Etiology:

- When allergens contact the respiratory mucosa, specific IgE antibody is produced in susceptible hosts
- Concentration of allergen in the ambient air correlates directly with the rhinitis symptoms

Epidemiology:

- Age at onset usually
- More common in those with a personal or family history of allergies/atopy

Clinical Features:

- · Nasal: obstruction with pruritus, sneezing
- Clear rhinorrhea (containing increased eosinophils)
- Itching of eyes with tearing
- Frontal headache and pressure
- Mucosa: swollen, pale, and "boggy"
- Seasonal (summer, spring, early autumn)
- -Pollens from trees
- -Lasts several weeks, disappears and recurs following year at same time
- Perennial
- Inhaled: house dust, wool, feathers, foods, tobacco, hair, and mold
- -Ingested: wheat, eggs, milk, and nuts
- Occurs intermittently for years with no pattern or may be constantly present

Complications:

- Chronic sinusitis/polyps
- Serous otitis media

Diagnosis:

- History
- Direct exam
- Allergy testing

Treatment:

- Education: identification and avoidance of allergen
- Nasal irrigation with saline
- Antihistamines (e.g. diphenhydramine, fexofenadine)
- Oral decongestants (e.g. pseudoephedrine, phenylpropanolamine) topical decongestant (may lead to rhinitis medicamentosa)
- Other topicals: steroids (fluticasone), disodium cromoglycate, antihistamines, and ipratropium bromide
- · Oral steroids if severe
- Desensitization by allergen immunotherapy