Acute and chronic rhinitis
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**Rhinitis**: This term refers to inflammation of the mucosa of the nasal fossae.

Owing to the continuity of the mucosa of the nasal mucosa and that of the sinuses, some degree of inflammation is often present in the latter at the same time, so constituting a rhinosinusitis.

When the inflammation of the sinus is primary or overshadows that of the nasal fossae, the condition is called sinusitis.
1. The common cold (coryza)

**Aetiology:**
A virus infection, which is conveyed by airborne droplet, usually complicated by secondary bacterial infection
Frontal sinuses with mucus

Inflammation causes swelling that blocks off the openings (ostia) of the sinuses, trapping mucus and air inside.
Pathology
Transient ischemia of the mucosa is followed by swelling, hyperemia and profuse secretion of clear seromucinous fluid. The rhinorrhea becomes mucopurulent later owing to the rapid growth of the resident flora of the nose, which appear to be activated by the virus infection.

The organisms founds includes
Streptococcus haemolyticus
Pneumococcus
Staphylococcus
Haemophilus influenza
Klebsiella pneumoniae
Branhamella catarrhalis
The pathogenic respiratory viruses include:
1. influenza viruses
2. picorna viruses comprising:
   . coxsackie virus
   . reonvirus
   . ECHO virus
   . rhinovirus ... most frequent cause of common cold
3. respiratory syncytial viruses (bronchitis in children only)
4. para influenza viruses (mainly in children)
5. Adenoviruses (mainly affecting pharynx)
Clinical features
Vary greatly. Four stages may distinguished:
1. *Ischmia stages*. After an incubation period of 1-3 days, a burning sensation is experienced in the nasopharynx. The nasal mucosa irritate, sneezing occurs and the patient feels chilled (shivering). Sense smell is altered or lost.
2. *Hyphaemic stage*. In a few hours, profuse rhinorrhoea and varying degrees of nasal obstruction ensue. Pyrexia is common.
3. *Stage of secondary infection*. As secondary infection and leucocytic invasion occur, the discharge becomes yellow or green.
4. *Stage of resolution*. Occurs in 5-10 days.
Differential diagnosis
1. allergic rhinitis and vasomotor rhinitis – rhinorrhoea spasmodic and the condition is apyrexia. The discharge remains clear and contains excess of eosinophils.
2. influenzal rhinitis in which the constitutional symptoms are much sever.
Complications
Secondary infection may spread throughout the mucosa and lymphatic tissue of the whole respiratory tract including the middle ear cleft.

Treatment

Prophylactic: contact with known cases must be avoided. Vaccines for prevention have not been widely effective.

Therapeutic

General treatment consist of
- rest and warmth. Ideally patient should stay in bed
- analgesics. Condeine, aspirin are of value
- pseudo-ephedrine by mouth relieves congestion
- antihistamine and vitamin c
- antibiotic should be reserved for the treatment of secondary infective complications
Local treatment

consist of:

. inhalation of steam comforting, Tinct. Benzoin Co or menthol may be added but it is not essential

. vasoconstrictors. In the form of drops or sprays give quick but temporary relief from the nasal obstruction. They should not be abused

2. acute rhinitis associated with influenza and the exanthemata

This differs little from that of a common cold, except for the presence of the associated disease of which it is usually a prodromal feature. It may however be very severe and even purulent. Complications are apt to follow and may be serious
Chronic rhinitis

1. non_specific chronic infective rhinitis
Several types are described

(a) simple chronic rhinitis

Etiology
Attacks of acute rhinitis is rapid succession and the rapid maintenance of the acute inflammatory condition by one or more of the many predisposing and contributory factors these include:

- neighboring infections such as: sinusitis, chronic tonsillitis, adenoids
- vasomotor rhinitis. the resultant obstruction predispose to chronic infection
- chronic irritation as from dust, smoke, tobacco, snuff and the abuse of therapeutic vasoconstrictors. Polluted atmosphere, as by overcrowding. sudden and extreme changes of the temperature. excessive dryness or humidity
- nasal obstruction leading to retention of discharge e.g. deviation of the septum and intranasal adhesions
- metabolic factors which includes imbalance of diet, as by excess of carbohydrate and by deficiency of vitamins. endocrine disorders, especially of the thyroid. lack of exercise and sunlight, alcoholic overindulgence, or gout.
Pathology

.chronic hyperemia of the nasal mucosa accompanied by inflammatory cellular infiltration and sometimes chronic oedema

.swelling of inferior turbinate due to engorgement of the cavernous spaces (sinusoids) in the submucosa, the epithelium tends to lose its cilia and the goblet cell increase. these changes' are reversible
Clinical features

- Nasal obstruction marked variable and usually alternate from side to side. On change of posture, the dependent side blocks as the inferior turbinate swells.
- Postnasal drip. A clear, viscid secretion trickles into the nasopharynx, to descend, this tends to become mucopurulent as infection increases.
- Nose—blowing
  - A blocked or heavy feeling in the nose. Common, with mild headache and mental apathy.
- Transient anosmia
Diagnosis
A history of recurrent attacks of acute rhinitis and the presence and of predisposing factors are suggestive. The soft swollen mucosa over the inferior turbinate pits with a probe and shrinks with 5% solution of cocaine. If morning sneezing is frequent the condition is likely to be associated with vasomotor rhinitis.
Treatment

general correction of any predisposing factors where possible e.g. tobacco, alcohol or contact with person suffering from cold change of habitat

Local a slightly alkaline nasal douche used night and morning to remove sticky mucus

Mild vasoconstrictors used as sprays, paints or drops, usually of 1-2% ephedrine preparations to which an antibiotic may be added. Such as treatment must not be prolonged. Topical steroids as spray or drop will help in some cases.

Treatment of any sinusitis or other adjacent infection
b) Hypertrophic rhinitis

Aetiology
The condition represents an advanced stage of simple chronic rhinitis, in which permanent hypertrophic changes have followed. The causes are similar, but it is frequently seen also in patients who have used topical decongestants in large quantities and/or for long periods (rhinitis medicamentosa).
Pathology

Permanent hypertrophic changes accompany the inflammatory oedema and cellular infiltration in all the constituent parts of the mucosa i.e. stroma, glands, blood vessels, and lymphatic tissue. The epithelium loses cilia and shows a tendency to squamous metaplasia. The mucosa becomes thick and nodular, especially at the extremities and free border of the nasal choana. Fibrosis can cause venous and lymphatic obstruction. If the resulting passive edema occurs in the situation where the mucosal stroma is loose, polypi form. Nasal polyposis, however, usually indicate an allergic or vasomotor origin of the rhinitis, in which the polyposis results from increases capillary permeability.
Clinical features
Similar to those of simple chronic rhinitis but are unremitting in character. Pitting of the firm mucosa with a probe and shrinkage with cocaine are less marked than in simple chronic rhinitis.
Treatment
As for simple chronic rhinitis, with the addition of limited reduction of the hypertrophied inferior turbinate and removal of any polypi. Topical decongestant preparation must be discouraged or discontinued. Reduction is achieved by:
- Electrocoagulation: by SMD or linear cauterization by galvanocautery
- Cryosurgical probe
- Surgical trimming of the hypertrophied free border
- Amputation of an posterior end end may be necessary
- Avulsion or total removal of the inferior turbinate may be performed, but persistent crusting can result
(c) Atrophic rhinitis

Definition
A chronic inflammation of the nasal mucosa, in which its various constituents undergo atrophy as a result of per arterial fibrosis and endarteritis of the terminal arterioles.
Aetiology
The exact etiology is not fully known but its incidence has markedly decrease in western Europe.
1. infection
2. undue patency of the nasal airway
3. endocrine disturbances
4. vitamins disturbances

Types
1. primary atrophic rhinitis (ozaena): possibly the sequel of rhinitis associated with exanthema in childhood the process may passed hypertrophic stage before becoming atrophic
2. secondary atrophic rhinitis: destruction of the nasal mucosa and subsequent healing are associated with fibrosis of the submucosa and metaplasia of the ciliated epithelium
   - it happen as result of
     - deviated septum
     - syphilis
     - lupus

Excessive operative procedure especially on the inferior turbinate
**Pathology**

Degeneration of the ciliated epithelium and seromucinous glands causes the formation of thick adherent crusts in the nose. These become secondarily infected with saprophytic organism. The bony structures of the turbinate atrophy, and the airway is widened. The sinuses may be small due to arrested pneumatization but are often normal. There appears to be a tendency to spontaneous recovery in later life in some cases.
Clinical feature
The conditions now seldom seen is bilateral and more common in female it appear about puberty
1. foul stench not noticed by the patient who is anosmic
2. epistaxis may follow separation of the crust
3. sensation of obstruction despite the unduly wide airway
4. similar atrophic changes may be seen in the pharynx and larynx

Differential diagnosis
1. sinusitis  2. syphilis
Treatment
Removal of crust is best achieved by syringing with warm isotonic solution
Glucose 25% in glycerin drops prevent adherence of fresh crust and inhibit saprophytic infection
Local or systemic antibiotic can be used initially as indicated by the sensitivity of the organism
Potassium iodide therapy and endocrine preparation such as stilboestrol locally or systemically
Surgical measures to reduce the caliber of the airway by submucosal insertion of graft, Teflon paste submucosal injection and to form adhesions between the septum and the infractured lateral nasal wall,
Moistening of the mucosa has been attempted by diverting Stenson's duct into the antrum
Complete surgical closure of the nostrils for periods of several months has been given striking improvement in the status of the mucous membrane (young operation)
(d) Rhinitis sicca
A rather ill defined crusting condition affecting the anterior third of the nasal cavity of patients who work in dusty surroundings. Periglandular fibrosis and metaplasia of the ciliated epithelium result in a viscid and stagnant mucus blanket which forms crust; these are not foetid but are sometimes bloodstained and may lead to a septal perforation. There is no generalized atrophic change or any increase in caliber of the airway.

**Treatment**
is by correction of occupational surrounding and lubrication by sprays, paints or ointment, applied to the affected area.
(a) Syphilis
Two forms
Congenital
Two forms are distinguished:

[Image of congenital syphilis]
1. Early form. up to the third month
A snuffle is the name given to the commonest nasal manifestation. this is a rhinitis which rapidly becomes purulent
Fissuring and crusting of the vestibule and lips are caused by the irritating discharge so that sucking may be prevented. Occasionally necrosis of bone and cartilage occurs.
2. Late form (third form to puberty) deformities similar to those in the tertiary stage of acquired syphilis result from gummatous infiltration of the nasal mucosa and subsequent destruction of the nasal framework. Other stigmata affecting the eyes, teeth, and ears.
Acquired
Has three stages

*primary stage*: onset 3-6 weeks after contagion
Chancre rare in the nose and appear a hard painless papule in the vestibule or on the adjacent septum it soon ulcerates to form a sore which has been linked to painless furuncle the submaxillary and preauricular glands disproportionately enlarged but painless

*Secondary stage*: onset 6-9 weeks after contagious
Persistent coryza the presenting symptoms in the nose. Mucus patches are rarely distinguishable on the nasal mucosa but they may be seen in the mouth and pharynx
**Tertiary stage**: onset usually 1-5 years after contagious a gumma of the nose is the commonest nasal manifestation of syphilis. Usually starting in the periosteum of the septum it soon cause perforation. When the nasal bone are involved the bridge sinks (saddleback deformity). Less often the nasal floor may be perforated by gumma while the lateral wall are rarely affected. The external nose may be also attacked. When the ala, columella and adjacent upper lip are destroyed.

Nasal obstruction and headache. The latter worse at night are the earliest symptoms.

Before ulceration a nasal gumma present as a non-pitting rubbery swelling which is reddish purple in colour may be diffuse or localized and is accompanied by mucoid rhinorrhoa.

After ulceration there is crusting and a foul bloodstained discharge the ulcer has a sloughy (washleather) base in which dead bone or fragment of necrotic cartilage may be detected.
saddle
nose
Diagnosis
Confirmation depends upon identification of the Treponema pallidum in smears from primary and secondary lesions. Serological tests are negative in the primary stage but positive in 90% of others.

Biopsy
Trial therapy
Treatment
General antisyphilitic treatment by systemic penicillin. Nasal toilet as by irrigation with warm hydrogen peroxide (10 vol). In snuffles suction followed by instillation of ephedrine solution may render suckling possible

Sequelae
Healing may result in vestibular stenosis, secondary atrophic rhinitis, perforation of the palate and septum and the deformities of the external nose
(d) Tuberculosis

Etiology
1. miliary spread from focus in the lung
2. fingernail inoculation, in the predispose tuberculous patient

Clinical features
1. tuberculoma occurs on the cartilaginous portion of the septum
2. ulceration and perforation of the septum follow and spread to the nasal floor and inferior turbinate. Nasal tuberculosis is much rarer than laryngeal tuberculosis
Treatment
For lung condition, and includes the use of long term antituberculous drugs (rifampicin, ethambutol, INAH, streptomycin, PAS)
(E) sarcoidosis
Histological appearances are similar to tuberculosis except for the absence of caseation. Mantoux test is negative. Kveim test is sometimes positive. The disease is widely distributed in the body, lung, salivary, and lymphatic glands, skin, and uveal tract being often affected in the nose. It may present as nodules on the septum and in the vestibules. Spontaneous recovery is usual.
Leprosy
An extremely chronic tropical infection due to mycobacterium leprae. Occurs as Asia, Africa and in Central and South America.

Pathology
M. leprae is conveyed to the septum by the fingernail. Initial coryza causes sneezing and discharge teeming with M. leprae. This is the infectious stage of the disease.

After an interval of up to 10 years, a nodular thickening develops at first on the septum. The nodules show a perivascular cellular infiltration of the tissue which includes vacuolated giant cells and bacilli in parallel bundles.

Untreated progressive ulceration goes on with subsequent destruction of the nose, palate and larynx may be involved.

Treatment
By rifampicin and clofazimine and dapsone for long period.