Inflammations of The Nasal Cavity

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Acute Inflammations:
A- Vestibulitis (diffuse, localized)
B- Rhinitis (non-specific, specific)

Chronic Inflammations:
A- Non-specific (atrophic, hypertrophic)
B- Specific (granulomas)
Fruncles of Nasal Vestibule

Acute localized infection in relation to a hair follicle in the skin lining of the vestibule caused by Staph. aureus

Predisposing factors: Nose picking, Diabetes.

Presentation: Throbbing pain with swelling of tip of nose, with edema and redness. If ruptured scanty purulent nasal discharge.

Complications: (dangerous area of face) Cavernous sinus thrombosis, facial cellulitis, septal abscess.

Treatment:
Antibiotics both systemic and topical
Analgesics
Instructions to avoid squeezing the area
If recurrent, diabetes is suspected
Nasal Vestibulitis

Diffuse inflammation of the skin lining of the nasal vestibule caused by pyogenic organisms.

Predisposing factors: following attacks of rhinorrhea, discharge and repeated rubbing of nose lead to skin maceration and fissuring.

Presentation: burning pain in vestibule area, with dryness, fissures and crustations.

Treatment:
- Treatment of the cause
- Local antibiotic ointments
Acute non-specific Rhinitis

Common Cold (coryza)

Acute non-specific viral rhinosinusitis caused by Rhinoviruses, and is usually followed by secondary bacterial infection. It is a form of upper respiratory tract infection in which nasal manifestations are more prominent.

Predisposing factors: bad housing, lowered immunity esp. in children exposure to cold air currents and local factors.

Pathology:
1- Stage of ischemia, vasoconstriction of nasal blood vessels
2- Stage of congestion, vasodilatation of blood vessels and excessive nasal secretions.
3- Stage of secondary bacterial infection (suppurative rhinitis)
4- Stage of resolution.
Presentation:
1- Stage of ischemia, burning sensation and dryness with sneezing, nasal mucosa is pale and dry.
2- Stage of congestion, mild constitutional symptoms with nasal obstruction and watery discharge. Mucosa is red and swollen
3- Stage of secondary bacterial infection, constitutional symptoms more severe, nasal obstruction more, discharge becomes thick mucoid, mucopurulent or purulent. Mucosa dusky red and edematous.
Complications:
Vestibulitis, spread of infection, permanent anosmia

Treatment:
Prophylactic. Avoid predisposing factors, avoid contact with patients, Vaccination
Curative. General (bed rest, ample fluids, analgesics, antipyretics, vit.C)
Specific (nasal decongestants, steam inhalation, antibiotics for secondary infection)
Influenza

Acute non-specific rhinitis caused by Influenza viruses (A,B,C). More severe than common cold, with complications.

Clinical picture: similar to common cold with following differences
1- Constitutional manifestations are more severe
2- Local manifestations are less prominent
3- Secondary bacterial infection is more common
4- Complications are more common (labyrinthitis or vestibular neuritis, meningitis, gastroenteritis, pericarditis)
5- Influenza may present in epidemic form (high morbidity and mortality)

Treatment: similar to that of common cold, vaccines are available especially to individuals with high risk
Acute Specific Rhinitis

Nasal Diphtheria

Acute specific rhinitis due to infection with C.diphtheria, secondary to faucial diphtheria and rarely primary.

Presentation:
Low grade fever with toxemia
Unilateral nasal obstruction with purulent nasal discharge, grayish dirty membrane

Complications and Treatment (as faucial diphtheria)
Chronic Non-specific Rhinitis

**Atrophic Rhinitis**

Chronic non-specific rhinitis characterized by progressive atrophy of nasal mucosa (glands, blood vessels, bony turbinates)

**Pathology:**
Ischemia due to endarteritis or periarteritis of nasal terminal arterioles.
Evaporization of water content of nasal secretions.
Dryness will initiate atrophic changes:-
Glands lead to diminished nasal secretions, nasal epithelium with destruction of cilia, turbinate bones.
Nose become wide or roomy.
Dry secretions form crusts, with secondary infection producing offensive smell or ozaena.
Primary Atrophic Rhinitis
a- Hormonal disturbances
b- Infection by Kl. Ozaenae in children
c- Autoimmunity
d- Autonomic imbalance with sympathetic overactivity
e- Deficiency of vit. A or iron

Secondary Atrophic Rhinitis
a- Rhinoscleroma
b- Syphilis
c- Septal deviation or perforation
d- Surgery
e- Chronic sinusitis
f- Radiation therapy
Presentation:
More common in females (atrophic), manifestations are bilateral
-Nasal discharge with crusts (greenish, grayish, offensive odor)
- Nasal obstruction inspite of having roomy nose (accumulation
  of crusts and functional as patient does not feel air current)
- Anosmia
- Epistaxis
- Sore throat.

The examiner notice foetid smell, nasal cavity is roomy with small
 turbinates. Mucosa is pale and dry , covered with crustations
**Treatment**

**Medical**
- Saline nasal washes for crusts separations
- Potassium iodide orally to stimulate glandular secretion
- Local estrogen
- Vit. A and iron
- Mucolytic agents
- Steam inhalation or humidifiers.

**Surgical**

Failure of medical treatment, surgery aims at:
1. Narrowing wide nasal cavity by medial displacement of lateral nasal wall or submucosal implantation of fat, bone, Teflon, cartilage
2. Temporary closure of anterior nares (one side at a time 6-24 month)
Chronic Specific Rhinitis

**Granulomas**

**Rhinoscleroma**

Chronic specific inflammation of URT due to infection by *Klebsiella Rhinoscleromatis* (Frisch bacillus). Usually starts at nose, then extends to: pharynx, larynx, trachea and nasolacrimal duct and sac.

**Pathology:** mode of transmission unknown. Passes into stages:

1- Stage of invasion; prolonged attack of acute non-specific rhinitis.
2- Second stage; may be atrophic or hypertrophic type or mixed

Atrophic reactions (2ry atrophic rhinitis)

Hypertrophic reactions (active nodular or diffuse granulomatous) bilateral submucosal hard nodules appear at mucocutaneous junction, then coalesce to form large masses that fill nasal cavity, may broaden nose. May extend to upper lip, pharynx, larynx or nasolacrimal ducts.
Microscopic examination: granulation tissue reaction characterized by high vascularity, Mikulicz cells (macrophages with large foamy vacuolated cytoplasm and small central nuclei, the G-ve bacillus is seen in these vacuoles and the presence of these cells is diagnostic), Russel bodies (degenerated plasma cells, appear red or round or oval bodies with no nuclei).

3- Stage of fibrosis (healed scleroma); heals by dense fibrous tissue leading to narrowing, or obliteration of nasal cavity with possible external deformity.
Clinical Picture
1- Stage of invasion simulates acute non specific rhinitis.
2- Atrophic stage simulates atrophic rhinitis may be some scattered granulations.
3- Active granulomatous stage; bilateral nasal obstruction with mucoid discharge. Examination may reveal external broadening, anterior rhinoscopy shows bilateral reddish masses filling nasal cavity covered by intact mm. Dyspnea and stridor if extends to larynx and epiphora if nasolacrimal duct is involved.
4- In healed stage; external nasal deformity, scarring and narrowing of nasal cavity

Sequelae
In active stage; upper lip deformity, dyspnea and stridor, epiphora. Fibrosis and scarring:- permanent nasal obst, ext nasal deformity. Pharyngeal affection with permanent shortening of soft palate. Laryngeal stenosis or tracheal stenosis. Malignant changes are rare.
Investigations
Biopsy
Bacterial cultures

Treatment
1- Medical (in atrophic and active stages)
   a- Antibiotics (Rifampicine 300mg/12hrs for 6-8 weeks, Ampicillin
       500mg/6hrs with sulpha cotrimexazole/12hrs 3-4 weeks, third generation
       cephalosporins, quinolones or according to C&S tests.
       (streptomycin is no longer used due to its cocheao-vestibular affection)
   b- Local measures (removal of crusts, moistening nasal
       preparations, Aureomycine oint locally for 1 year)
2- Surgery (to treat sequelae of fibrosis)
   Recanalization, Rhinoplasty, Dacrycystorhinostomy
3- Radiation therapy (not used any more for fear of malignancy)
Chronic specific rhinitis caused by *Treponema pallidum*. Types: Congenital (transplacental infection) and acquired.

Pathology:
1- Acquired syphilis may be primary (very rare to affect nose, chancre is painless raised red papule with lymphadenitis), secondary (multisystem, mucus patches may ulcerate, generalized skin rash and lymphadenopathy) and tertiary (diffuse lesion as 2ry atrophic rhinitis, localized gumma painless rubbery swellings ulcerate to from punched out ulcers with bone and destruction; posterior perforation of nasal septum, depressed nasal bridge, oro-nasal fistula, CSF rhinorrhea).

2- Congenital may be early form (during first 3 months of life, resembles 2ry acquired with nasal snuffles and fissuring) and late form (after third year of life, resembles tertiary diffuse reaction or gumma).
Investigations
1- Serological tests: diagnostic in 2ry and 3ry
   a- F.T.A & VDRL (screening)
   b- Treponema pallidum immobilization test T.P.I
2- Smears from chancre or mucus pateches
3- Biopsy from 3ry

Treatment
1- Penicillin is drug of choice.
2- Local treatment for 2ry atrophic.
3- Plastic and reconstructive surgery for nasal, septal and palatal deformities and perforations
**Lupus Vulgaris**
Chronic specific form of T.B infection that affects skin and mucus membranes, caused by attenuated tubercle bacilli and the mode of infection by direct innoculation (nose picking).

**Pathology** discrete nodules at mucocutaneous junction, ulcerates forming small shallow undermined ulcers, it can affect cartilage with destruction of septal cartilage resulting in anterior septal perforations. Healing follows with extensive fibrosis to vestibule, nasal tip and surrounding skin.

**Clinical Picture**
Prolonged nasal obstruction, discharge and crustations. Bilateral discrete firm nodules at mucocutaneous junction of vestibule and skin of butterfly area of face. Nodules appear as apple jelly when pressed with glass silde. Anterior perforation of nasal septum, marked scarring of skin of face and nose
Investigations
1- Biopsy is diagnostic.
2- Bacteriological examination T.B.
3- Tuberculin test is not useful.

Treatment
1- Specific antituberculous drugs (triple combination of rifampicine I.N.H. and ethambutol for 9 months)
2- Vit D
3- Surgical reconstruction.