

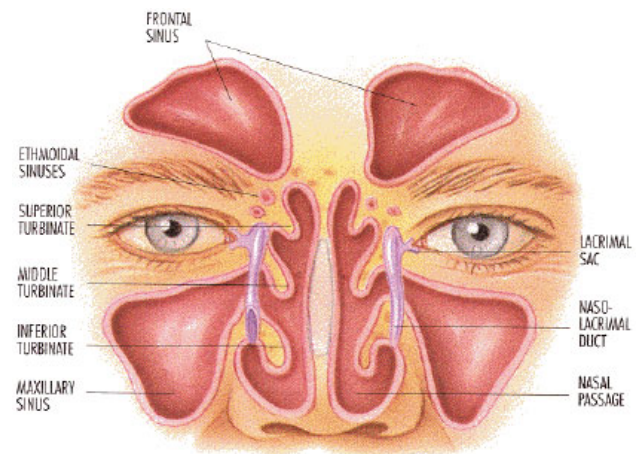
NOSE III

-Sinusitis-

- Nasal sinuses: air-filled cavities within the bones.
- Sinusitis: inflammation of the mucoperiosteal lining of paranasal sinuses.
- Sinusitis is very important. Sinuses close to the eyes and base of skull.
- Pediatricians, primary care physicians and ENT physicians all need to know how to diagnose and manage sinusitis.
- It is actually called *Rhinosinusitis* –not sinusitis- because the sinuses are in continuity with the nose, lined by the same structures, and affected by the same process.
- If a disease happens in the sinus → usually affects the nose as well.
- If the cause was dental → affect only the sinuses.
- Unable to differentiate clinically; because they are within one cavity and lined by same epithelium.
- Isolated sinusitis is rare. (only one sinus, or with no nose involvement).

ANATOMY

- There are 4 pairs of paranasal sinuses: **①** Frontal. **②** Ethmoid. **③** Sphenoid. **④** Maxillary.
- In some references, earliest sinus to develop is the *maxillary*, and *ethmoidal* in others. They're present at birth.
- Sphenoid and frontal appear by 3rd year.
- Latest sinus to develop is the frontal.
- The *ethmoidal* sinuses are separated by basal lamella that separate posterior from anterior ethmoid & it's an important surgical landmark.
- *Lamina papyracea* & *cribriform palate* are important structures because they are the routes of infection.



Lamina papyracea: lateral part of ethmoid bone and the medial wall of the orbital wall; the area separating the ethmoid from the orbit.

- **Openings:**
 1. *Frontal, anterior & middle ethmoid cells, and maxillary antrum* open in the middle meatus –also called *osteomeatal complex area*- the major area of sinusitis.
 - So if middle meatus is diseased or blocked → all sinuses are affected except *posterior ethmoid* and *sphenoid*.

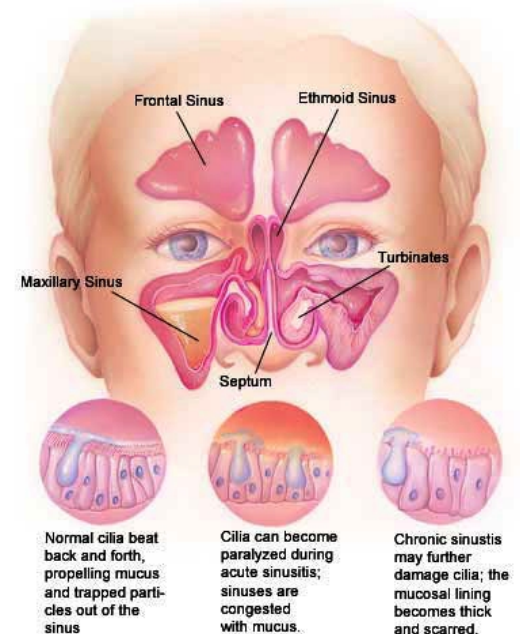
[Important structures in osteomeatal complex are:

- ① *Uncinate process*, ② *Infundibulum*, ③ *Semilunaris hiatus*, and ④ *Ethmoidal bulla*.]
2. *Posterior ethmoid* opens in the superior meatus.
3. *Sphenoid* opens in the sphenoidal recess.
4. *Nasolacrimal duct* opens in the inferior meatus.

- *Concha Bullosa*: aeration of middle turbinate (a distention of the turbinate bone due to aeration -like a cyst-, it's a normal variation)

PATHOPHYSIOLOGY

- Nasal mucosa lined by (respiratory-like) pseudostratified columnar ciliated epithelium (The same lining of nose and trachiobronchial tree). Except the anterior 1 cm of nose (nasal vestibule) lined by stratified squamous epithelium (skin-like).
- Normal nasal function depends on:
 - ❶ Patent open ostia.
 - ❷ Normal ciliary function
 - ❸ Normal quality of mucus secretion.
 (i.e. normal irritation and normal drainage, otherwise we end up with a blind cavity).
- Mucus blanket is in two layers: a superficial viscous layer and an underlying serous layer.
- Mucus drainage: 1. By cilia out of the nose.
2. Swallowed then taken care of by gastric acid.
- Cilia and mucosa beat in a specific pattern (from lower part of sinus going toward the natural ostium) (in *frontal sinuses*, from medial to lateral until it gets to frontoethmoidal recess area)
- Cilia beat in the serous layer, moving circularly the blanket bypass the artificial opening towards the natural nasal ostia. (In the nose they are beating toward the nasopharynx).
- They beat propelling posteriorly into the nasopharynx then either swallowed or spit out.
- Usually diseases of the cilia (e.g. infection, smoking, cystic fibrosis and immotile ciliary syndrome) present with rhinosinusitis.
- Most important pathological process in the disease is obstruction natural ostia.
- Artificial opening –in inferior sinus- does not drain the sinuses, it only aerate them. So restoration of the normal function of the sinuses is the treatment rather than drainage.



[Obstruction of the meatus and the natural ostium of the sinuses → Hypo-oxygenation
→ ciliary dysfunction → poor mucous quality → retention of secretions → good media
for microbes to grow]

CAUSES AND PREDISPOSING FACTORS

1. **Inflammatory** (*URTI & Allergy*) → commonest cause.
 - UTRI:
 - The most common.
 - Viral.
 - Usually resolves within 2 days.
 - 3% end with rhinosinusitis → mostly in patients with anatomical predisposing factor.
 - Allergy:
 - Another common etiology predisposing to the inflammatory rhinosinusitis.
2. **Local Factors:** can impair ciliary function (cold air → “states” the epithelium. Dry air → desiccates the blanket). Sinus barotruma (Swimming, flying, and diving).
3. **Anatomical Factors:** blocking the ostia (e.g. polyps “in most cases obstructing their nasal airway”, tumors, nasal deviation “rare”, foreign bodies, large adenoid “particularly in children → retention of secretions”, turbinate hypertrophy, osteomeatal complex obstruction, cleft palate, choanal atresia, and rhinitis).
4. **Bad hygiene and overcrowding.**
5. **Low general condition** (debilitation, diabetes mellitus, and immunodeficiency).
6. **Gastroesophageal Reflex**
7. **Cystic Fibrosis**
8. **Kartegner’s Syndrome** (there is situs inversa).
9. **Immotile Cilia Syndrome** (no situs inversa).

DEFINITIONS

- Time can differentiate between bacterial and viral. Viral takes 7-10 days to resolve, if symptoms persist more *or* worsen after 5 days → bacterial.
- Based on duration; acute, sub acute, and chronic in all sinusitis, tonsillitis, and otitis media. But it’s not an accurate classification cause we could have *acute on chronic* or *recalcitrant* (resistant).

Acute Rhinosinusitis: Infection that resolves within *3 weeks*. (Usually within 2 days)
Divided into severe and non-severe forms.

Recurrent Subacute Rhinosinusitis: Infection lasting between *3 weeks* to *3 months*.

Chronic Rhinosinusitis: Infection lasting more than *3 months*.
Low-grade symptoms and signs.

[Treat medically unless *acute case with complications* or *chronic*]

CLINICAL PRESENTATION

[History and physical examination are vital to proper diagnose]

1. Viral URI (URI symptoms takes up to 10 days so unable to differentiate at first).
2. Serous rhinosinusitis. May be mucopurulent → WBCs “pus”, can be is severe viral too not only bacterial.
3. Nasal congestion and cough.
4. Nighttime cough, which may linger.
5. Low grade fever (mostly pediatric)
6. Malaise.
7. Headache.

Acute non-severe rhinosinusitis:

- Persistent cold symptoms over 10 days.
 - ① Rhinorrhea (anytype), ② cough (dry or wet) which worse at night, ③ low grade fever, ④ fetid breath, ⑤ painless peri-orbital swelling (morning), and ⑥ facial pain (rare), dental pain and headache.

Acute Severe Rhinosinusitis:

- Usually after 10 days.
 - ① Purulent or copious rhinorrhea. (Nasal or post-nasal purulent discharge)
 - ② high fever.
 - ③ peri-orbital swelling.
 - ④ facial pain (sphenoid sinuses involvement).
 - ⑤ headache (severe when it involves the *sphenoid* and *frontal* sinuses. Periodicity is important. Old books: appears mostly in the morning and disappear in afternoon. Best description: aggravated when supine and when leaning forward “sjoed”)
 - ⑥ dental pain (can be a cause o sinusitis).

DIAGNOSIS

- Acute rhinosinusitis is usually *not* seen by ENT but seen in primary care and pediatric.
- Chronic rhinosinusitis usually seen in ENT.

A. **History.** [2 majors *or* 1 major and 2 minor]

- Major factors:
 1. Facial pain or pressure or congestion.
 2. Nasal obstruction.
 3. Nasal/postnasal discharge. Purulent.
 4. Hyposmia (reduced smell) or anosmia.
 5. Fever.
 6. Purulency in nasal cavity on examination.

- **Minor factors:**
 1. Headache. (if very severe, it's a major factor especially in *sphenoid* and *frontal*)
 2. Cough.
 3. Fatigue.
 4. Halitosis.
 5. Dental pain.
 6. Ear pain or pressure.
- B. **Physical Examination.** [Pus in nasal cavity highly indicative; if pus is seen coming from the nose its an enough clue of rhinosinusitis]
1. **Anterior Rhinoscopy with Otoscope** (to see polyp or discharge). [Using different specula to see anterior half of the nose]
 2. **Oropharynx and Nasopharynx.** [Looking for polyps or pus]
 3. **Tenderness over sinuses.**
[Mostly with acute *frontal* sinusitis → can be touched]
Because: it's a confined space, adjacent to the brain, and because of the nerve endings.
 4. **Pin orbital edema and discoloration.**
 5. **Endoscopy:** “*examination of choice in rhinology*”
Passing through inferior or middle meatus into nasopharynx.
Could be misdiagnosed if not visualized all the way through.
(From the anterior nares to the posterior choanae).
Depending on what you're viewing; *Naso-pharyngeal Laryngoscopy*.
- **Rigid Endoscope:** “preferred” give better visualization showing adenoid size and nasopharynx but needs cooperation from patient because it could injure the nose.
 - **Flexible Endoscope:** children and mentally restarted. “non-cooperative patients”
Gives two-dimensional images only, if 3D needed, use microscope or naked eye.



NON-SPECIFIC:

- C. Colored mucopurulent discharge (pus does not always indicate bacterial infection)
- D. Peri-orbital swelling and facial tenderness.
- E. Transillumination (no value)
- F. Ultrasonography (little value)

INVESTIGATIONS

[Do not investigate in acute and subacute as they are treated empirically.
Only investigate in *chronic* and if *complications* appear]

A. RADIOGRAGHY

1. Traditional X-ray views:

- Water's view [occipitomental view] → Maxillary sinus. “mostly frontal sinuses”.
- Caldwell view [occipitofrontal view] → Ethmoidal & Frontal sinuses and *don't* see the maxillary.
- Lateral view → Frontal, Maxillary, Sphenoid and *Adenoid*.
- Submentovertical view → needs positioning, quite difficult.
(disadvantages: low sensitivity & specificity)

2. Computed Tomography: **“V.IMP”**

- *Gold Standard*. In chronic and acute with complications.
- Coronal “perpendicular to hard palate”, axial “parallel to hard palate” and sagittal “reformatted” cuts. Nowadays only axial obtained from CT “multiplaner” and then reformat coronal and sagittal from the computer.
- Only way to diagnose *Concha Bullosa*.
- Planning surgery (Some cases the cribriform is lower than usual and CT is the way to know)
- Failed medical management.
- Some cases the cribriform is lower than usual and CT is the way to know.
- Indications:
 - i. Clinically unresponsive to medical treatment.
 - ii. Immunosuppressed patients.
 - iii. Severe symptoms or signs.
 - iv. Life threatening complications.

(Disadvantages: under estimate small rhinosinusitis)

3. MRI:

- With orbital or cranial complications.
- Shows *soft tissue* and we compare it to the other side.
- Does *not* show the bones.

B. SINUS ASPIRATE

- Swab and culture from middle meatus *not* from nasal cavity.
- Middle meatus → best site for aspiration.
- Useless if aspirated from nose cavity → there could be contamination.
- Indications are similar to CT's.
- Geriatric and children not responding to treatment.
- Needs cooperative patients (usually SMTA --)

C. MICROBIOLOGY

- Most common microorganisms: ① *Streptococcus pneumoniae*, ② *Branhamella* (*Moraxella*) *catarrhalis*, ③ nontypeable *H.influenzae*, and ④ *Strep. pyogens*. → the four gram +ve diplococci in all acute ENT infections.
- We give an antibiotic covering these four.
- Sometimes viruses, anaerobes, staphylococcus, and other microbes.
- In chronic: usually polymicrobial. Gram -ve (*bacteroids*, *klebsiella*, and anaerobes)
- Normal flora in the sinus – controversy.
[Some are sterile]

MEDICAL MANAGEMENT

- Antibiotic for 10-14 days.
 - *Penicillin* “antibiotic of choice”: to cover gram +ve diplococci.
 - 1st and 3rd generation cephalosporin.
- Decongestant: topical or systemic.
 - Topical not used because of rebound effect and there’s a chance of addiction. E.g. Atropine.
 - Systemic: no rebound effect after discontinuation.
- Topical steroids: if allergy on top of sinusitis.
- Symptomatic treatment: headache → analgesics.
- Treat underlying cause: allergy...etc;
- 40-60% sinusitis resolve (AOM)
- Historically – aspiration and irrigation.
- Antibiotic – Viral URI are common increasing numbers of drug resistant bacteria.
- 35% of *S.pneumoniae* → Penicillin resistant.
- 16% of *S. pneumoniae* → Penicillin intermediate.
- Rapid cures prevent complications and chronic sinusitis, and sterilize sinus.

Cephalosporins:
 1st generation → +ve
 2nd generation → +ve and -ve
 3rd generation → -ve

ACUTE NON-SEVERE RHINOSINUSITIS (no AHX)

- Amoxicillin (44-96 up to one day)
- 16 to 14 days ...
- PCM- allergic may ...
- Antihistamine: dry mucosal sensation & aeration.
- Isotonic: nasal drops, ... irrigation and steam inhalation.
- Topical or systemic decongestant.
- Nasal steroid.

[If one course of antibiotic fails after 2 weeks duration, start on another course for 2 weeks and observe, if fails, start a third course of 2 weeks and then if it fails we investigate “CT” and interfere surgically as the patient developed a chronic course]

RECALCITRANT ‘RECURRENT” RHINOSINUSITIS

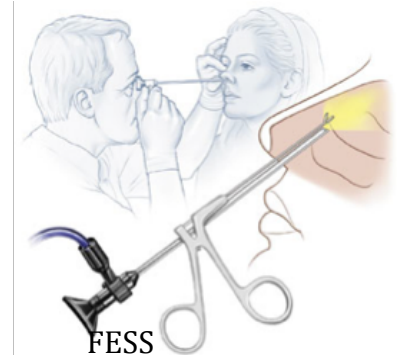
- You have to look for underlying cause:
 1. Allergy.
 2. Immunodeficiency.
 3. Cystic fibrosis.
 4. Ciliary dyskinesia syndrome.
 5. Gastroesophageal disease
- Repeat treatment 2-3 times over 2-3 months. If it doesn’t resolve then it goes into chronic → obtain a CT.

SURGICAL MANAGEMENT

If medical fails.. Surgery is the last option.

In the past: 1.Trephine frontal sinus. 2.Going through canine fossa cleaning the maxillary sinus but not addressing other sinuses.

1. Caldwell-Luc: damages dentition.
2. Inferior antrostomy:
 - Goes against proven ciliary outflow. Possible for ciliary dyskinesia/CF.
3. Functional Endoscopic Sinus Surgery (FESS):
 - Usually using a camera.
 - Functional → restores functions.
 - Aim of the surgery: drain and aerate → functions restored.
 - Types:
 1. With CT guidance. In difficult or complicated cases. Showing how far we are from optic nerve and base of the skull. Very common.
 2. Powered instrument and shaver: to cut and suck.
 3. Mini-FESS: aerate and drain *only*. Not reliable nowadays.
 - Controversial, difficult, too radical (AOM), reversible changes on CT.
 - Excellent results: 71% normal at 1 year, meta analysis –more accurate- 89% success with 0.6% complications.
 - Usually maxillary antrostomy & anterior ethmoidectomy.
 - Absolute indications: Complete, polyp., intracranial or complications., and fungal sinusitis.
 - Possible indications:
 1. Persistent chronic rhino sinusitis that fails ... and after ... of systemic disease.
 2. With rhinosinusitis.
 - **Post-operative care:**
 1. Sinus package.
 2. Oral antibiotic: for at least 2 weeks.
 3. Aggressive nasal hygiene.
 - **Complications:**
 1. Blindness. Diplopia.
 2. Orbital fat herniation.
 3. CSF leak “if we’re hitting base of the skull”
 4. Epiphora: overflow of tears.
 5. Retro-orbital hematoma.



COMPLICATIONS OF SEVERE ILLNESS

- Complications could be: orbital, cranial, and extra cranial.
- Routes of orbital spread:
 1. Direct. (via the sinus ostium: acute rhinitis, swimming in polluted water. Neglected nasal pack)
 2. Arterial.
 3. Venous. (They are valveless so infection can go antegrade and retrograde easily)
 4. Lymphatic.
 5. Dental (in maxillary)

ORBITAL COMPLICATIONS

Five grades: “Chandler’s classification” for acute infection.

1. **[Grade I]: Inflammatory edema; lid edema otherwise normal**
 - Proptosis, epiphora, vision loss “even without the previous”, preseptal edema...etc;
 - Usually referred from ophthalmology.
 - We have to investigate: CT, MRI “if indicated”
 - Periorbital inflammatory edema.
 - Obstruction of venous channel
 - No vision loss.
 - No EDM limitation.

2. **[Grade II]: Orbital cellulitis: diffuse edema**
 - Infection involving whole orbit.
 - Sometimes it’s hard to differentiate cellulitis from abscess.
 - Compare sides.
 - Orbital cellulitis with edema, chemosis, proptosis and pain.
 - Ophthalmoplegia may occur due to edema or inflammation of the muscles.
 - No visual loss.
 - No abscess.

3. **[Grade III]: Subperiosteal abscess: usually seen near lamina paprycea.**
 - Subperiosteal abscess: Extending from ethmoid to the periosteal region. (Air bubbles seen in subperiosteal region and elevation of periosteum compared to the other side)
 - Subperiosteal abscess.
 - Globe displaced laterally and downward.
 - Orbital cellulitis with decreased extra ocular muscle movement.
 - Vision deterioration.

4. **[Grade IV]: Orbital abscess: collection within orbits.**
 - Orbital abscess: pus → drain.
 - Severe proptosis and chemosis.
 - Usually no global displacement.
 - Ophthalmoplegia.
 - Visual loss (13%) → due to ischemia and neurosis.

5. **[Grade V]: Cavernous sinus thrombosis:**
 - **Bilateral:** infection going from one to the other because they’re valveless.
 - Usually results from retrograde transmission through valveless veins leading to the cavernous sinus.
 - CN II , IV, and VI. “rare”
 - Meningitis.
 - Heralded by bilateral orbital involvement, progressive chemosis, T 10SF.
 - High mortality rate.
 - Tx: drainage and IV antibiotics.
 - Heparin is controversial.

[A new grading for both acute and chronic complications. **Grade 1:** Anatomical disturbances. **Grade 2:** Functional disturbances. **Grade 3:** Orbital infection. **Grade 4:** Visual impairment.]

MANAGEMENT OF COMPLICATIONS

1. History and physical Examination
2. Ophthalmology consultation.
3. IV multiple antibiotic (cefotaxime *or* ceftriaxone “3rd generation covering gram -ve” + Clindamycine “covering gram +ve and anaerobes”) + metronidazole “flagyl”)
4. CT scans.
5. Surgery (Indications for surgical drainage: ①Progressive orbital cellulitis. ②Symptoms that do not resolve. ③Abscess. ④Loss of visual acuity.)
6. FESS, if can't be done → External drainage and frontal sinus trephining.

[Know what sinus is involved]

[Abscess treated with → surgical drainage and IV antibiotic] [Neurosurgery, ophthalmology, ID]

INTRACRANIAL COMPLICATIONS:

1. Meningitis: common in children.
 2. Subdural abscess:
 - 1/3 – 2/3 of subdural abscess in children caused by sinusitis.
 - Neck rigidity → first symptom.
 - Neurosurgery has to manage the intracranial pressure.
 - Surgical drainage of the abscess.
 3. Epidural abscess.
 4. Cerebral abscess.
- Indication for MRI. “Superior”

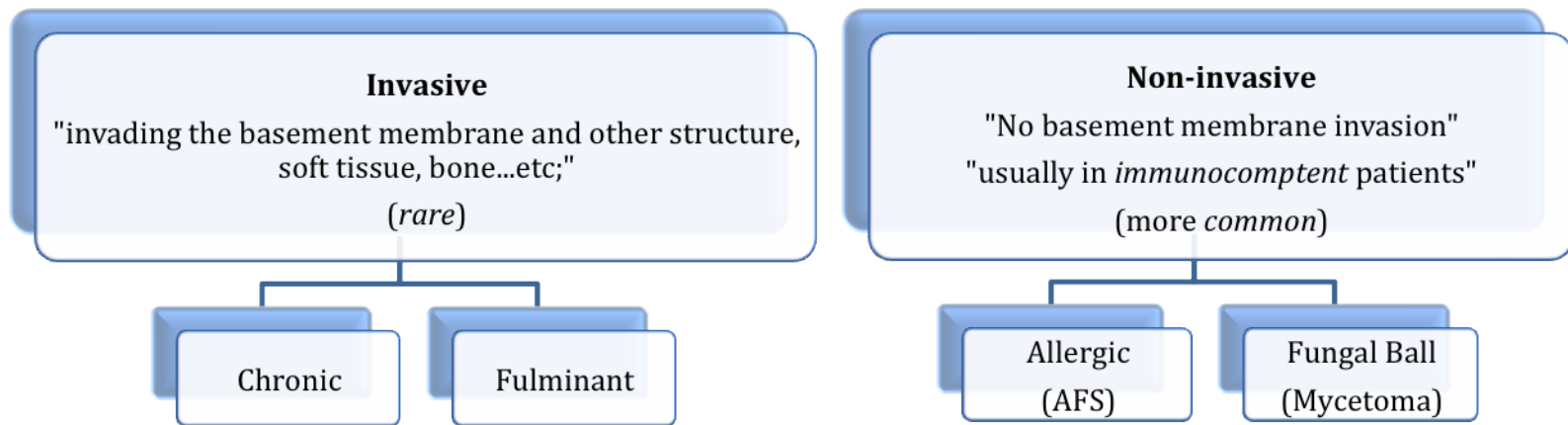
- When frontal, ethmoid, and sphenoid reach the base of the skull they could cause intracranial complications.
- Sometimes intracranial-extradural extension; respecting the dura.

EXTRACRANIAL COMPLICATIONS (MUCOCELE)

- Mucocoeles are chronic, cystic lesions of the sinuses lined by pseudostratified epithelium.
- Mostly found out incidentally, frequently noted on routine CT scan.
- Etiology is debated: 30% *idiopathic* and 70% *iatrogenic* “surgeon causing the obstruction leading to mucocoele”. (rather due to obstruction of the ostia or to simple obstruction of minor salivary gland).
- Expand *slowly*, often requiring many years.
- Maxillary sinuses. Sphenoid and ethmoid mucocoele less common.
- Frontal sinuses mucocoele → proptosis and even blindness (important to recognize)
- Causing pressure on adjacent structures.
 - Can cause: osteitis or osteomyelitis → swelling and protrusion of frontal region.
 - Supraorbital fissure syndrome: pressure on the apex of the orbit.
 - Orbital apex syndrome: collection of cranial deficits.
 - Sinocatenoid fistula. ??
 - Dacryocystitis: inflammation of the nasolacrimal sac due to obstruction of nasolacrimal duct.
- Seen with vortex, headaches and deep nasal pain.
- No treatment required unless near natural ostia.
- Therapy involves obliteration of the sinus.
- Treatment is controversial: wide cyst drainage into the nasal vault is common.

Osteitis: bone infection.
Osteomyelitis: bone and bone marrow infection.

FUNGAL SINUSITIS



1. NON-INVASIVE ALLERGIC FUNGAL SINUSITIS (AFS)

- Very common. Seen on daily basics in clinics.
- Hypersensitivity type I to fungi antigen (↑ IgE). An immune reaction rather than infection.
- Expansile: affects the brain and meninges by extending and expansion.
- Mimic chronic sinusitis
(Differentiation: non-expansile, by CT, experience “with proptosis → AFS”)
- Allergic Bronchopulmonary Aspergillosis (ABPA): hypersensitivity to aspergillosis – aspergillus fumigatus- mimics AFS. But they don’t need surgery to diagnose.
- Radiological: unilateral (sometimes bilateral), expansile, calcification, heavy metal depositions.
- Associated with: proptosis, erosion of the sinus wall, and sometimes septal deviation → due to expansion.
- Most common fungus: *Aspergillus flavus*.
- Criteria for diagnosis: not definite unless operate and histopathology. (debris and hyphae)
 - a. Testing (culture or staining) or history of fungal atopy.
 - b. Nasal polyposis. “most of the time”
 - c. Mucin.
 - d. CT demonstrating heterogeneous hyperdense material in the sinus cavity. (calcifications)
 - e. MRI → void (low) signal.
 - f. No histological evidence of fungal invasion on tissue biopsy.
- Treatment:
 - FESS in all cases.
 - As in chronic sinusitis + steroids.
 - Steroids pre-op (2 weeks) → to shrink polyps, ↓ edema and minimize bleeding.
 - Post- op (3 months) → suppress immunity, as it’s a recurrent systemic disease.
 - Antifungal (ketoconazole or itraconazole): used in recurrent cases that do not respond to steroids and surgery → oral “systemic” antifungal → partial response sometimes. “controversial”
- Recurrence is high. Even though it’s aerated and drained.

DD_x of unilateral nasal mass:

1. AFS.
2. Antrochoanal polyp.
3. Inverted papilloma.
4. Tumors (squamous mostly)

Squamous cell is the most common in all ENT malignancy even in middle ear and UR due to metaplasia.

2. NON-INVASIVE FUNGAL BALL (MYCETOMA)

- A fungal ball sitting in the paranasal sinus.
- Usually in the maxillary sinus.
- Treatment: Surgical excision followed by antifungal sometimes.

3. INVASIVE FULMINANT FUNGAL SINUSITIS

- Usually in *immunocompromised* (leukemia, AIDS, and cytotoxic drugs)
- Caused by mucormycosis (angioinvasive mold) → attack the blood vessels → tissue ischemia.
- Tx: debridement of all non-viable tissue.

4. INVASIVE CHRONIC FUNGAL SINUSITIS

- Immunocompetent
- Tx: surgical debridement.

5. INVASIVE ACUTE FUNGAL SINUSITIS

- Uncommon.
- Immunocompromised. (Mostly seen in *diabetic ketoacidosis*)
- Fatal, 90% mortality.
 1. Aspergillosis (most common)
 2. Mucormycosis: patient comes with little superficial cellulitis and orbital involvement but the disease is aggressive.
 3. Candidiasis.
 4. Histoplasmosis.
 5. Coccidioidomycosis “valley fever”
- Initially seen as engorgement in the turbinate → ischemia, necrosis, and black charring of the tissue.
- Fungus invades the vascular channels → block blood vessels → hemorrhagic ischemia and necrosis.
- Require high index of suspicion. (patients with *diabetic ketoacidosis* → mucormycosis)
- Diagnoses: biopsy and culture.
- Treatment:
 - Treat underlying cause.
 - Parenteral amphotericin B.
 - Radical surgical debridement.