



Nose III-IV

Objectives:

- Acute & chronic sinusitis (causes, clinical & management)
- Fungal sinusitis (in brief)
- Complication: sinusitis (classification, management & with special attention to)
- Orbital complications (investigation & general treatment)
- Radiology illustration
- Diseases-nasal septum (DNS)
- Epistaxis (causes, clinical mngt)
- Turbinate hypertrophy
- Nasal operations (FESS, septoplasty, turbinate surgery) in short

Resources: Female Slides , 437A

Done by: Abdulhakim Bin Onayq, Hadeel Awartani, Alanoud Almufarrej, Mashel Alkahtani

Edited by: Reem Alqarni, Esraa Alnazzawi

Revised by: Rotana Khateeb

[Color index: **Important** | **Notes** | Extra]

Types of Sinusitis

Acute sinusitis: **less than 4 weeks**

- a sudden onset of cold like symptoms such as runny, stuffy nose and facial pain that does not go away after 10 to 14 days. Acute sinusitis typically lasts 4 weeks or less.
- **Acute sinusitis has flu like symptoms that last longer than an influenza and new symptoms appear**

subacute: **between acute and chronic**

- An inflammation lasting 4 to 8 weeks

chronic sinusitis: **more than 8-12 weeks**

- a condition characterized by sinus inflammation symptoms lasting 8 weeks or longer.

Recurrent sinusitis:

- several attacks within a year.
- **Recurrent sinusitis is not common so you have to intervene.**

Acute Sinusitis

- Inflammation of the mucosal lining of the nose (**same as sinus lining; recently called rhinosinusitis**) and paranasal sinuses. Affects 35 million patients in the US annually, with 16 millions office visits. Women are more affected in some studies (2:1, ?dealing with children)

● Pathophysiology:

Three general factors: Obstruction of sinus drainage, Ciliary impairment, Altered quality or quantity of the mucus.

Microbiology	
Acute Viral	Acute Bacteria most common
Rhinoviruses, influenza A and B, Para-influenza, corona viruses, RSV, adenoviruses, enteroviruses	Streptococcus pneumoniae, haemophilus influenza, moraxella catarrhalis.

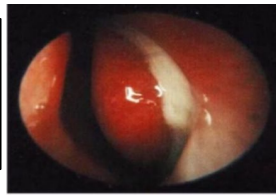
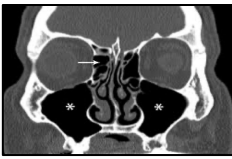
- **Clinical PODS:** Hallmark of acute sinusitis (to differentiate it from URTI or flu) : Nasal congestion + **headache** + **facial pain and tenderness** + post-nasal drip + **purulent nasal discharge** (most URTI cause runny nose with watery discharge meanwhile in sinusitis its yellowish or greenish)
 - **Pain/Pressure:** **facial** (frontal(**forehead pain**), periorbital(**or between eyes if ethmoidal**), cheek(**maxillary**), dental(**maxillary**)).
 - **Nasal Obstruction:** unilateral vs. bilateral, complete vs. partial,
 - **Discharge:** anterior (**secretions coming out from nose**) vs. posterior (**post nasal discharge, feeling like something is stuck in throat**), thick vs. thin, clear vs. mucopurulent. **Purulent nasal discharge.**
 - Smell: anosmia vs. hyposmia.
 - Duration: more than 10 days (or worsening after initial improvement) but less than 3 months.
 - Fever, fatigue, **headache** which gets worse when leaning forward (patient will say when i pray or try to get something from the floor i feel like my head is heavy), hoarseness due to postnasal drip causing patient to cough which will affect vocal cords eventually , halitosis (**bad breath**), cough mainly with **children** and constitutional symptoms.
 - Ear symptoms: clicking of the ear. (**eustachian tube closes due to infection or enlargement of turbinates**)
 - Visual and neurological symptoms (complications).
 - Dental issues/pain (as a cause of sinusitis) due to **maxillary nerve**
 - History of immune suppression

- **Physical Exam:**

- Fever
- Nasal tenderness and erythema around the nostrils
- Facial erythema or tenderness
- Mucosal oedema, erythema, purulent discharge
- Causative issues: septal deviation, hypertrophied inferior turbinates
- Clinical exam: Anterior Rhinoscopy , nasal endoscopy
- Nasal endoscopy or anterior rhinoscopy is used **to confirm dx** by the presence of pus or discharge coming from the sinus
- if you don't have an endoscope you can use a nasal speculum to examine the nose
- type of bacteria confirmed by taking a swab



Discharge (pus) is seen at middle meatus which is a hallmark of acute sinusitis. I can say 100% sure sinusitis



Discharge coming from maxillary sinus

● Investigations:

- CBC: for WBC, ESR in a sick patient
- Sinus x-rays: not used now, very low yield.
- Sinonasal culture: not done as routine, only if no response to ATB and suspicion of unusual or resistant organisms. endoscopic guided
- CT scan Sinuses: mostly requested when suspension complications.

● Treatment Acute sinusitis: Usually medical only

- **Antibiotics:** The most common organism is bacterial. Most common bacterial organism in adults: strept. pneumonia unlike children
- when to give Abx? if you found pus yellowish in color or symptoms getting worse or complications
 - First line: amoxil, clarithromycin or azithromycin. no need to know names
 - Second line: Amoxi-clav, flouroquinolone
- Supportive:
 - Intranasal corticosteroids (nasonex)
 - Analgesia (non- narcotic)
 - sinus irrigation
 - decongestant (local and systemic) atropine used for 3 days works by decongesting the mucosa so the pus comes out (to decrease congestion and open ostia of sinuses)
 - hydration
 - smoking cessation, etc.
- Most patients improve **spontaneously** or after a course of conservative management. **specially if viral**
- If **no response**, or develop recurrent symptoms should be considered more aggressive medical management such as: **anti-histamines, prolonged course antibiotics, decongestants, nasal steroids or vasoconstrictors.**
- **Surgery** is reserved for patients who fail 3 to 5 months of medical management or have complications. **Or if it became chronic or if it was recurrent.**

Chronic Sinusitis

One of the most common disease with direct impact on the quality of life of the patient. Persistent inflammation of the nose and paranasal cavities that lasts more than 12 weeks. or 8 weeks

we have to intervene and do surgery if it became chronic B.C it affects life

● **Pathophysiology:**

- Persistence of infection
- Allergy or other immunologic disorder
- Intrinsic factors of the airway
- Superantigens
- Fungi that induce and sustain eosinophilic inflammation
- Metabolic abnormalities such as ASA sensitivity

● **Etiology:** Normal infection that continuous due to (predisposing factors for sinusitis to become chronic):

- **Obstruction**(so secretion will collect in sinus, stagnate and get infected) : tumors, trauma, tubes, anatomical (nasal septum deviation, enlarged turbinate), etc persistent from acute
- **Allergy and atopy** (asthma, ASA sensitivity, allergic rhinitis) more tendency, they have hyper-reactive immune system and and their mucosa will always be congested closing the sinus opening.
- **Defects in ciliary clearance** (PCD) pseudocolumnar epithelial defect (congenital defects in cilia, cilia continually moves nasal secretions into nasopharynx, if there's a defect in ciliary clearance it will lead to stagnation of secretions in the sinus) and quality of mucus (CF) cystic fibrosis in which secretion is very thick so they always present with sinusitis
- **Hormonal** (puberty and pregnancy) due to decreased immunity
- **Irritant** (smoking, pollutant, acid reflux)
- **Immune** deficiency (AIDS, DM, renal transplant, immunosuppressant medications) cartilaginous syndrome= chronic sinusitis + chronic discharge bronchitis. in immune deficiency fungal sinusitis is our concern because some types are fatal and must be diagnosed within few hours.
- **Systemic** (Wegener, Churg-Strauss syndrome, sarcoidosis)
- **Dental** rarely oroantral fistula. some patients might have their root of last tooth inside the sinus which will cause fistula between mouth and maxillary sinus if the tooth was extracted, so anything can go inside the sinus such as food causing chronic sinusitis.
- **Most common bacterial:** Staph Aureus, Coagulase negative staph, Pseudomonas, Bacteroides and other anaerobes.

● Types of chronic sinusitis:

Chronic Sinusitis VERY IMP	
Chronic Rhinosinusitis (CRS)	Allergic Fungal Sinusitis Mainly related to immunological status so fungus stagnate in sinus
<ul style="list-style-type: none">● with nasal polyposis: CRSwNP● without nasal polyposis: CRSwoNP	

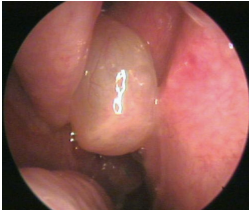
● Clinical PODS: Pain+ facial smell+congestion

- symptoms are less severe than acute, **NO fever**
- Pain/Pressure: facial (frontal, periorbital, cheek, dental).
- Obstruction: unilateral vs. bilateral, complete vs. partial.
- Discharge: anterior vs. posterior **usually posterior**, thick vs. thin, clear vs. muco-purulent **usually**
- **Smell**: anosmia **can't smell** vs. hyposmia **decrease smell**(i can only smell **strong odors**).
- Duration: More than 3 months.
- Fatigue, Malaise, Halitosis, Cough
- Ear symptoms
- Visual and neurological symptoms (complications)
- Dental issues (as a cause of sinusitis)
- History of immune suppression
- Exacerbation of asthma
- Need to know about previous medical and surgical treatment, their effectiveness and duration of benefits
- Exposure to allergens
- **Usually in chronic sinusitis all sinuses are involved**

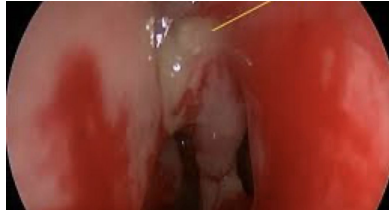
● Physical exam:

- Facial erythema or tenderness
- Mucosal oedema, erythema, purulent discharge (**we don't usually see pus because its not acute unless if the polyp stayed for a long time and obstructed the sinus**), polyps (**single big, or multiple small**)
- Causative issues: septal deviation, hypertrophied inferior **or middle** turbinates

- **Dental exam** (specially if you're suspecting oro-antral fistula) for tenderness and dental hygiene due to maxillary sinusitis
- Orbital, cranial nerves examination when complication is suspected



Nasal polyp in middle meatus



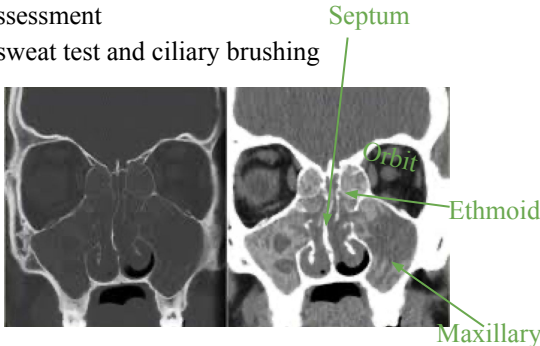
Purulent discharge

Note: Any patient with unilateral sinusitis is a red flag for tumor

*Acute sinusitis is a clinical diagnosis not a radiological

Investigations:

- **Endoscopic Examination:** to assess erythema, edema, discharge, polyps, anatomical variations
- **CBC:** for WBC and eosinophilia
- Sinonasal swab for culture
- **CT scan:** most important to establish diagnosis, to assess severity and extent of disease, to r/o complications and for surgical planning and mapping.
- **MRI:** in cases of orbital and intracranial complications
- **Other tests:** IgE and other immunoglobulins, ESR, CRP, RF, ANA, C-ANCA, P-ANCA, lymphocytes subtypes, HIV serology.
- Allergy assessment
- Chloride sweat test and ciliary brushing



Mostly fungal because it has areas of hypodensity and areas of hyperdensity which is characteristic of a fungal infection

Treatment chronic sinusitis: Medical and surgical

- we start by medical unless its not responding to treatment or you suspect malignancy or other complications

- **Local treatment:**
 - o Intra-nasal corticosteroid sprays: mainstay Rx **most imp** because **antiinflammatory**
 - o Sinus rinses: **most important** **water and salt sprays** that **clean the nose and remove secretions**
 - o Others: no evidence or recommendation against
- **Systemic treatment:**
 - o Po Steroids: short term (5-15 days, different regimens) **only if big polyp** **presurgery**. **not responding** to intra nasal steroids
 - o Po ATB: **If polyp** with signs of infection such as pus
 - First line: amoxil, clarithromycin or azithromycin.
 - Second line: Amoxi-clav, fluoroquinolone.
- **Surgical (FESS)**
 - o Functional Endoscopic Sinus Surgery **opening sinus and clean it**

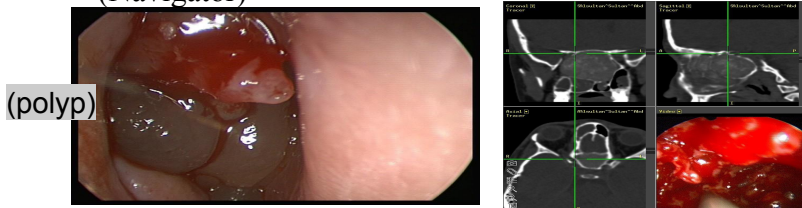
● Surgical Management:

- FESS : **gold standard for chronic rhinosinusitis, acute when there is complication**



- Computer Assisted Surgery (Navigator)

Done with pt. has intracranial or intraorbital.

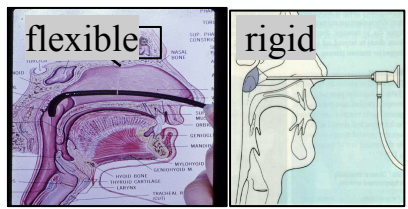


- Balloon Sino-plasty
 - new procedure, expensive
 - good for fronto-ethmoid recess



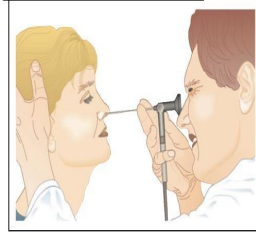
- **Endoscopic Examination:**

- Rhinoscopy
- Endoscopy (pic) →
 - rigid (poorly tolerated)
 - flexible (best way)

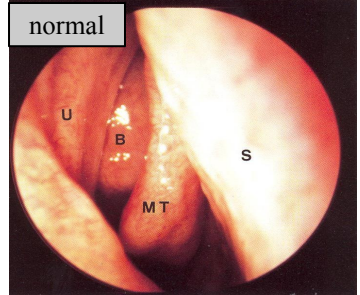


rhinoscopy

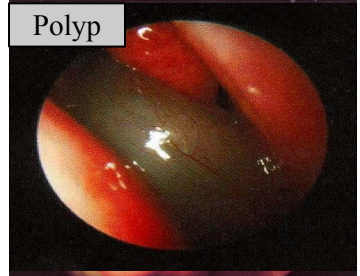
endoscopy



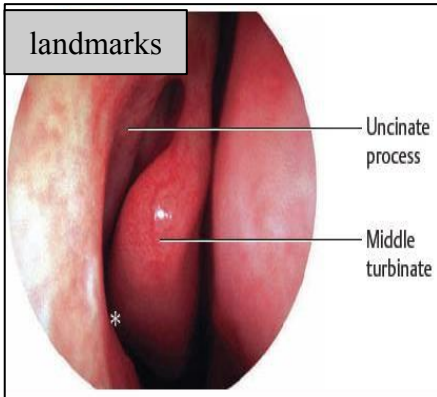
normal



Polyp



landmarks



Uncinat process

Middle turbinate

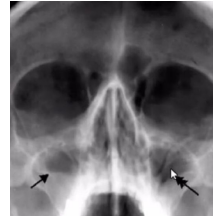
U:uncinat process
B:bullae ethmoidalis
MT: middle turbinate
S: septum

Radiology

- **Clinical indication for diagnostic imaging**

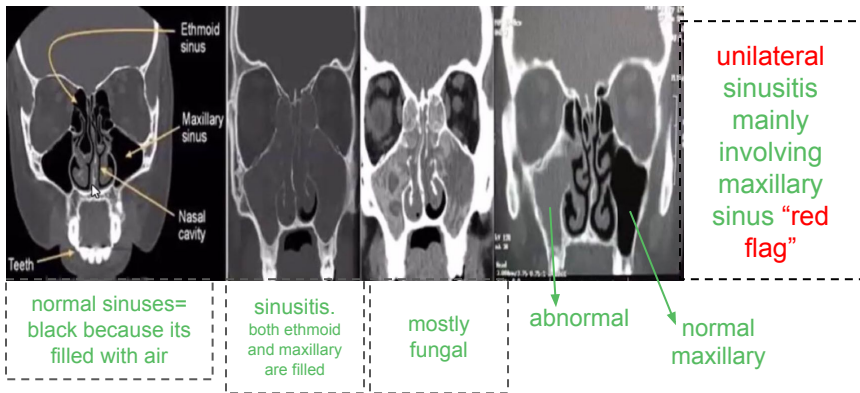
- **Not indicated** in acute, uncomplicated rhinosinusitis or to confirm the resolution of the infection.
- The goal of sinus imaging is to visualize the sinus to determine if there is an underlying **anatomical disorder** contributing to the **persistence or recurrence** of symptom.
- **imaging in case of chronic** is used to know which sinus is inflamed and if there is complications, or in case of recurrence. we don't need imaging in acute unless there is anatomical disorder due to trauma, or severe deviation

- **Plain (X-ray) sinus films (rarely indicated) not done anymore**
- Plain films don't differentiate the etiology (infectious vs. non infectious)
- Correlate poorly with clinical events
- Over 80% of children with persistent respiratory symptoms have abnormal findings on plain films.
- There is poor correlation between x-ray and C.T. scan findings.



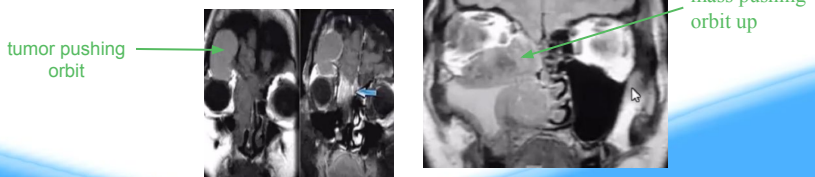
- **C.T. scan indications**

- recurrent or chronic sinus disease when surgery is being considered.
- Complicated rhinosinusitis with signs of extension beyond the bony sinus.
- Bony changes of chronic inflammation from osteitis.
- Recurrent or persistent mucoceles.
- Large polyps on physical exam. or abnormal looking polyp
- sinus tumors/malignancy. **unilateral nasal polyp is a red flag**



- **MRI of the sinuses**

- To evaluate suspected intracranial or orbital involvement of complicated rhinosinusitis. (if you're suspecting extension of tumor to brain or orbit.)
- Mapping of sinonasal neoplasms.



Sinusitis Complications

COMPLICATIONS OF Rhino SINUSITIS

Table 5. Complications of Acute Sinusitis

Bony	Orbital
Osteomyelitis	Cavernous sinus thrombosis
Pott's puffy tumor	Inflammatory edema and erythema (preseptal cellulitis)
Intracranial	Orbital abscess
Cavernous sinus thrombosis	Orbital cellulitis
Epidural abscess	Subperiosteal abscess
Intracranial abscess	
Meningitis	
Subdural abscess	
Superior sagittal sinus thrombosis	

Information from references 7 and 9.

VERY IMP no need to need to know all detail

FYI: pott's puffy tumor is when someone has acute frontal sinusitis leading to pus formation so it breaks the weak area which is the anterior cover of the sinus so pus collects under skin of forehead



Three main complications:

- Orbital
- Cranial
- Extracranial

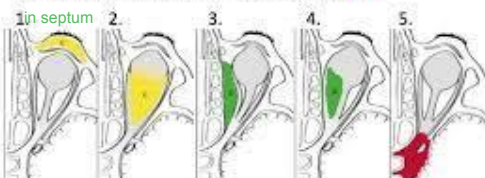
● Orbital Complications: IMP

- The **frontal, maxillary, ethmoid** and **sphenoid** sinuses sit immediately **above, below, between** and **behind** the eyes respectively.
- Infection of any of the sinuses may spread to the orbit causing complications from mild inflammation of the eyelid to abscess with possible blindness. **Intra-orbital spread of sinusitis**
- Chandler classification "Orbital Complication": next slide

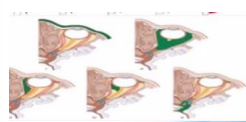
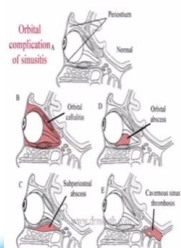
VERY IMP

Orbital Complications of Sinusitis

1. Periorbital (Pre-Septal) cellulitis (c)
2. Orbital (Post-septal) cellulitis (c)
3. Subperiosteal Abscess (a)
4. Orbital Abscess (a)
5. Cavernous Sinus Thrombophlebitis (a)



- **Preseptal cellulitis**: lid edema otherwise normal
- **Orbital cellulitis**: diffuse edema
- Subperiosteal abscess: usually seen near lamina papyracea
- Orbital abscess: collection within orbit
- Cavernous sinus thrombosis : bilateral



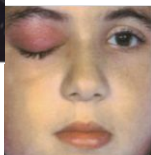
● Stage I: For Acute

- The infection out of the socket of the eye في الجفن (septum)
- Periorbital pre-septal inflammatory edema (cellulitis)
- Obstruction of venous channels
- No vision loss
- No EOM limitation “Extra-ocular muscle”
- common in children
- treated medically



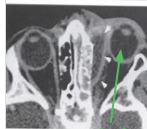
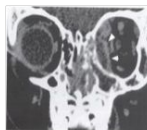
- Stage II:

- Passed through septum to orbit
- Orbital cellulitis with edema, chemosis, proptosis bulging due to inflammation that pushes the orbit, pain
- No abscess so we can still treat it with antibiotics
- Ophthalmoplegia may occur due to edema (not due to muscle its due to lid swelling) or spasm
- **No visual loss.** Visual loss requires emergency surgery



● Stage III:

- Subperiosteal abscess (under periosteum) starts pushing muscles of eye movement, so eye movement will be affected. also pushes the nerve a little but mainly muscles
- Globe displaced laterally or downward
- Orbital cellulitis present with decreased EOM
- **Vision decreased.** depends on size of abscess
- we put him on ABx and assess vision, we have to drain it surgically if it didn't respond to ABx



eye pushed outward

● Stage IV:

- Orbital abscess can lead to blindness B.C it starts compressing orbit nerve & can go to cavernous sinus.
- Severe proptosis (bulging of eye) and chemosis
- Usually no globe displacement
- Ophthalmoplegia present can't move eye muscles
- visual loss (13%) due to ischemia or neuritis due to the compression of blood vessels by the abscess



● Stage V:

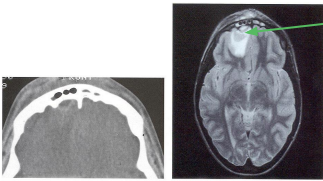
- Cavernous sinus thrombosis usually results from retrograde transmission through valveless veins leading to the cavernous sinus through optic nerve



- starts unilateral then becomes bilateral
- fixation of orbit (no movement of eye at all)
- Progressive symptoms
- proptosis and fixation
- CN II, IV, VI, III, V(v1 and v2). abducent is first to be affected
- Meningitis and thrombosis
- High mortality 50% with antibiotics

● Intracranial Complications

- The **frontal, ethmoid and sphenoid** sinuses are separated from the intracranial cavity by a layer of bone.
- If infection spread it may cause meningitis or brain abscess.
- Meningitis **Common in Children**
- Subdural or Epidural Abscess
- Cerebral Abscess:
Neurosurgery, Ophthalmology, ID(infectious diseases) (they should be involved)



brain Abscess as a result of untreated frontal sinusitis

● Treatment of acute complications:

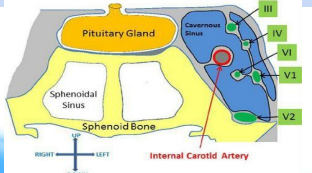
- Admit the patient
- IV antibiotics: 3rd Generation of Cephalosporins + Clindamycin
- Abscess incision & drainage and Surgery of the primary site
- Consultation of the Related Speciality

● CT scan PNS (paranasal sinuses)



subperiosteal abscess

● Cavernous sinus thrombosis



Sinusitis Complications (cont.)

❖ Mucocoeles

- Mucocoeles are **chronic**, cystic lesions of the sinuses lined by pseudostratified epithelium
- **Expand slowly**, often requiring many years
- Etiology Either due to obstruction of ostia or to simple obstruction of minor salivary gland
- 30% are idiopathic

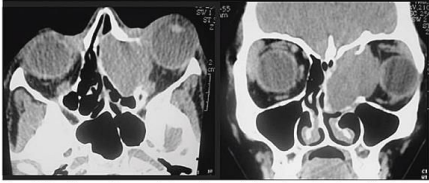
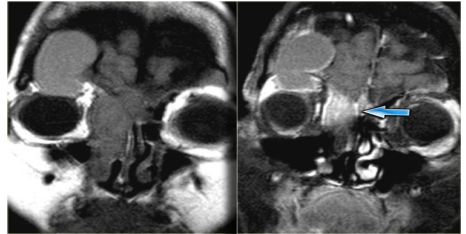


Figure 2. Nasal fossa and paranasal sinuses CT scan at axial and coronal sections evidencing image of left frontoethmoidal region with displacement of ocular globe on the left (proptosis).



❖ other complications:

- **Osteitis:** diagnose initially with technetium bone scan (osteoblastic activity) and gallium bone scan (inflammation), follow with gallium scans; Rx: parenteral antibiotics, surgical debridement, sinus surgery
- **Pott's Puffy Tumor:** frontal bone osteomyelitis, soft doughy swelling of forehead, high risk of intracranial extension; Rx: parenteral antibiotics, trephination, may require surgical debridement
- **Superior Orbital Fissure Syndrome:** fixed globe, dilated pupil (CN III, IV, VI), ptosis, hypesthesia of upper eyelid (CN V1); Rx: urgent surgical decompression
- **Orbital Apex Syndrome:** similar to Superior Orbital Fissure Syndrome with added involvement of optic nerve (papilledema, vision changes)
- **Sinocutaneous Fistula:** usually begins as a frontal osteomyelitis

Fungal Sinusitis

In the objectives, but female doctor didn't mention it

❖ Invasive fungal sinusitis:

- Presence of fungal hyphae within the mucosa, submucosa, bone, or blood vessels of the paranasal sinuses
- Acute Invasive Fungal Sinusitis (cause: mucormycosis)
- Chronic Invasive Fungal Sinusitis
- Chronic Granulomatous Invasive Fungal Sinusitis

❖ Non-invasive fungal sinusitis:

- Absence of fungal hyphae within the mucosa and other structures of the paranasal sinuses.
- Allergic Fungal Sinusitis (will be detailed in the next slide)
- Fungus Ball (fungus Mycetoma)

❖ Invasive:

- Presence of fungal hyphae within the mucosa, submucosa, bone, or blood vessels of the paranasal sinuses:
 - Acute Invasive Fungal Sinusitis - Most common patients in general are immunocompromised, usually due to diabetes, cancer, HIV, organ transplantation or using systemic or intranasal glucocorticoids
 - Chronic Invasive Fungal Sinusitis usually seen in patients who are less immunocompromised with a time course greater than 12 weeks
 - Chronic Granulomatous Invasive Fungal Sinusitis
 - Mucormycosis is encountered in dust and soil and enters through the respiratory tract
 - Ketoacidosis predisposes to mucormycosis, as the fungus thrives in acidic environments
 - Initially seen as engorgement of turbinates, followed by ischemia and necrosis of the turbinates and adjacent nose
 - The fungus invades vascular channels and causes hemorrhagic ischemia and necrosis
 - Frequently fatal. 90% mortality in immunocompromised
 - Patients with acute invasive fungal sinusitis are usually hospitalized and are very sick with fever, cough, nasal discharge, headache, and mental status changes.
 - Signs and symptoms include dark ulcers on the septum, turbinates, or palate. In the late stages, signs and symptoms of cavernous sinus thrombosis are present.

Treatment of acute invasive fungal sinusitis: Initial systemic antifungal treatment after surgical debridement. High doses of amphotericin B (1--1.5 mg/kg/d) are recommended followed by oral itraconazole, correction of underlying immunosuppression.

Treatment of chronic invasive fungal sinusitis: Surgical treatment is mandatory. Initiate medical treatment with systemic antifungals once invasion is diagnosed.

Fungal Sinusitis (cont.)

❖ Allergic fungal Sinusitis:

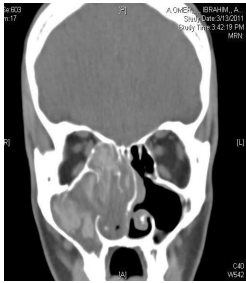
- Nasal obstruction
- Allergic rhinitis, or chronic sinusitis
 - Nasal congestion, Purulent rhinorrhea, Post-Nasal Drainage, or Headaches
- Patients are atopic
 - Unresponsive to antihistamines, Intranasal Corticosteroids, and prior immunotherapy
- Patients are always immunocompetent
- 5-10% of chronic rhinosinusitis patients actually cases of AFS(allergic fungal sinusitis)
- Two thirds of patients report a history of allergic rhinitis
- 90% of patients demonstrate elevated specific IgE to one or more fungal antigens.
- 50% of patients in a series by Manning et al had asthma.
- No linkage to aspirin sensitivity has been established.

❖ Examination:

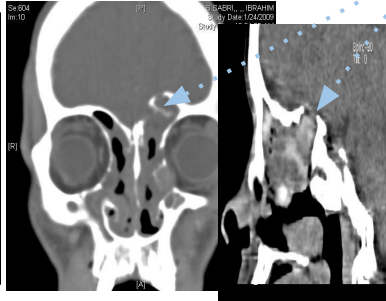
- **findings are typically broad**
 - intranasal inflammation and polyposis
- **facial polymorphism:**
 - ptosis
 - telecanthus
 - malar flattening
 - more often in children
- **orbital features**
 - proptosis occurring over a long period, no diplopia.
 - visual loss, from ophthalmic nerve compression or inflammatory process

Fungal Sinusitis (cont.)

❖ CT scan

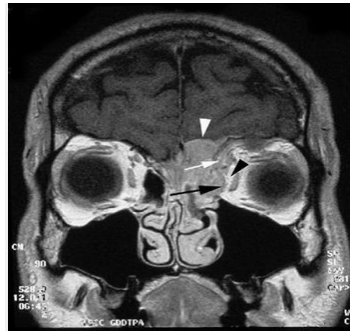
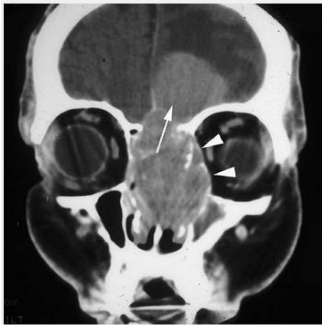


pathognomonic
-calcification
-heterogeneity
-expansion

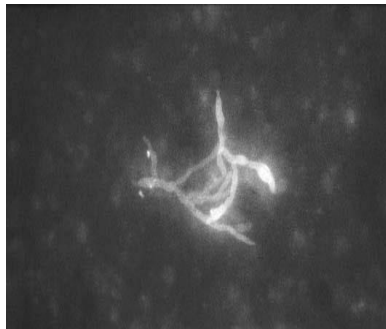
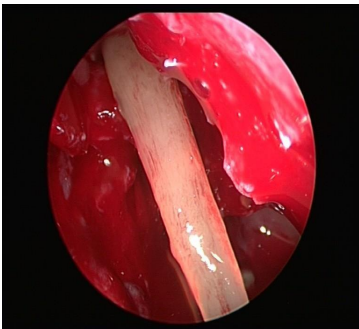


invasion

❖ Intracranial extension



❖ Mucin & fungal stain



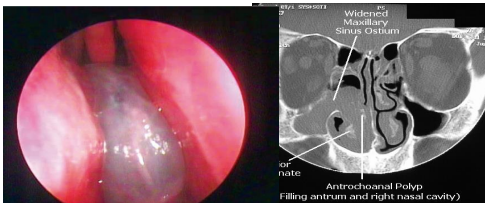
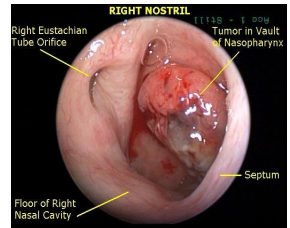
Fungal Sinusitis (cont.)

❖ Treatment of allergic fungal sinusitis

- The treatment of choice
 - Endoscopic debridement (FESS)
 - a perioperative short course of **steroids**.
- **Postoperative** mold containing **immunotherapy** is a promising therapeutic advance in limiting recurrence.
- The role of systemic antifungal therapy is inadequately studied. (just mentioned to have a complete discussion)
 - Itraconazole orally is well tolerated and effective in vitro against common causes of AFS

Unilateral Nasal Mass (mentioned here because its a presentation of allergic fungal sinusitis)

- Allergic Fungal Sinusitis (most common)
- Antrochoanal Polyp
- Inverted Papilloma
- Carcinoma



Fungal Sinusitis (cont.)

❖ Invasive fungal sinusitis

- Mucormycosis is encountered in dust and soil and enters through the respiratory tract
- Ketoacidosis predisposes to mucormycosis, as the fungus thrives in acidic environments
- Initially seen as engorgement of turbinates, followed by ischemia and necrosis of the turbinates and adjacent nose
- The fungus invades vascular channels and causes hemorrhagic ischemia and necrosis
- Frequently fatal. 90% mortality in immunocompromised

❖ Treatment

- Treated with radical surgical debridement
- Amphotericin B
- Correction of underlying immunosuppression

- **Anatomy**

- Frontal sinus is absent (aplastic) in most patients
- All the sinuses drain into the lateral wall, except? -sphenoid (drain into the sphenoidal recess)
- middle meatus is also called “osteomeatal complex”
- medial orbital wall is called lamina papyracea

- **Management**

- Rhinosinusitis is caused mostly by gram+ → use penicillin
 - or 2nd generation cephalosporin
- FESS surgery is minimally invasive
- if both medical and surgical treatment failed, what's the next step?
 - use biological treatment

- **Complications**

- head and neck venous system is valveless
 - infections can easily spread
- infections of membranous bones which have no bone marrow is called osteitis (not osteomyelitis)

- **Fungal sinusitis**

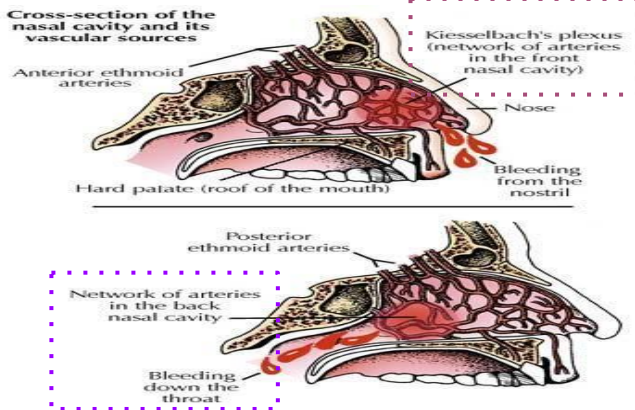
- how to differentiate invasive and non-invasive?
 - by histopathology
 - (if the basement membrane is intact-> non-invasive)
- acute invasive vs chronic invasive fungal sinusitis
 - chronic: no blood invasion, no necrosis
- treatment of invasive fungal sinusitis is radical surgical (debridement until we find a fresh blood)
 - +amphotericin B

Epistaxis

these factors and;
-heating and humidification properties
-vasculature runs just under mucosa
all contribute to nose bleed

- ❖ **Nasal blood supply** Blood vessels in face are valveless
 - internal and external carotid arteries
 - many arterial and venous anastomoses
 - **Kiesselbach's plexus (Little's area)** most common area of epistaxis because it is exposed to air any irritation will cause bleeding in anterior septum (Causes anterior nose bleeding)
 - **Woodruff's plexus** in posterior septum (Causes posterior nose bleeding)
 - Nose is highly vascular

❖ vascular anatomy of the medial and lateral walls



- ★ Kiesselbach's plexus/Little's area:
 1. Anterior Ethmoid (Oph).
 2. Superior Labial A (Facial).
 3. Sphenopalatine A (IMAX).
 4. Greater Palatine (IMAX)

- ★ Woodruff's Plexus:
 - Sphenopalatine A (IMAX).
 - IMAX = Internal Maxillary Art.Details of each part is not imp utst know two areas

❖ Types of nosebleeds Imp to know the location to manage

- **Anterior (little's area):** 90% of cases resolve spontaneously
 - Most common in younger population
 - Usually due to nasal mucosal dryness
 - Usually controlled with conservative measures
- **Posterior (vicinity of sphenopalatine foramen):**
 - Usually occurs in older population
 - HTN and systemic diseases are common contributing factors
 - Significant bleeding in posterior pharynx
 - More challenging to control

Epistaxis (cont.)

❖ Local causes of epistaxis

Nasal trauma (nose picking, foreign bodies, forceful nose blowing). Acute or chronic

bleeding polyp of the septum or lateral nasal wall (inverted papilloma)

Chemical irritants/ environment: pollutants, irritants, allergens

medications (topical), INCS, cocaine

Inflammation of the nose and sinuses/ Drying of the nasal mucosa from low humidity

tumors of the nasopharynx especially nasopharyngeal angiofibroma, juvenile nasopharyngeal carcinoma, neoplasms of the nose or sinuses

idiopathic

vascular malformation

Allergic, chronic or infectious rhinitis.

Deviation of nasal septum or septal perforation

❖ Systemic causes of epistaxis

Usually cause general bleeding not only to the nose

systemic arterial hypertension, atherosclerosis

anticoagulants (aspirin)

endocrine causes: pregnancy, pheochromocytoma

Blood diseases and coagulopathies such as: Thrombocytopenia, ITP, Leukemia, Hemophilia (bleeding disorders)

Familial hereditary hemorrhagic telangiectasia

hepatic diseases, aging

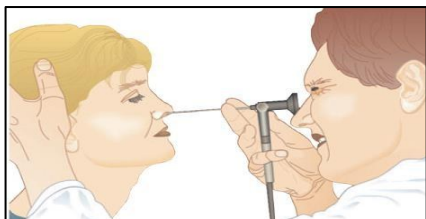
Epistaxis (cont.)

- Patient history

- Previous bleeding episodes
- Frequency, quantity, unilateral vs. bilateral, dizziness or LOC,
- Nasal trauma
- Family history of bleeding like hereditary telangiectasia
- Hypertension - current medications and how tightly controlled
- Hepatic diseases
- Use of anticoagulants
- Other medical conditions - DM, CAD, Low Hgb, etc.

- physical exam

- ABC's, blood pressure, and vital signs
- Apply direct pressure to external nose to decrease bleeding
- Use vasoconstricting spray mixed with tetracaine in a 1:1 ratio for topical anesthesia
- Identify the bleeding source (Anterior rhinoscopy and endoscopy)



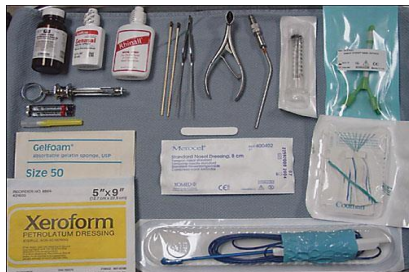
endoscopy



anterior rhinoscopy

- Equipment

- Protective equipment - gloves, safety goggles
- Headlight if available
- Nasal Speculum
- Suction
- forceps
- Tongue depressor
- Vasoconstricting agent
- Topical anesthetic



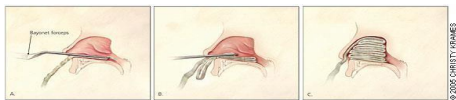
Epistaxis (cont.)

❖ Treatment of Anterior Epistaxis

- Localized digital pressure by pinching the cartilaginous part of nose and leaning forward so patient doesn't swallow blood for minimum of 5-10 minutes, If takes longer go to hospital
- Silver nitrate cautery (Chemical cauterization) If pressure doesn't stop
- Anterior nasal packing for refractory epistaxis bleed after cautery. or if extensive
- PostNasal Packing
- Arterial Ligation (Maxillary, Ethmoid, External Carotid) if everything failed
- Arterial Embolization
- Collagen Absorbable Hemostat or other topical coagulant

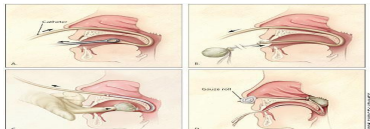
Anterior Nasal Packs If heavy bleeding or nonstop

- Formed expandable sponges are very effective
- Available in many shapes, sizes and some are impregnated with antibacterial agents



PostNasal Packs

- If anterior packing did not control the bleeding.
- Can be done using a foley catheter.
- Do if anterior packing doesn't stop bleed



- Duration of Packing Placement

- Actual duration will vary according to the patient's particular needs.
- Typically, anterior pack at least 24-48 hours (after we control the bleeding), sometimes longer.
- Posterior pack may need to remain for 48-72 hours.
- If a balloon pack is used, advised tapered deflation of balloons.
- If patient still bleeds after packing then ligate large vessels (maxillary or external carotid) or embolization

- Patients with Nasal Packing

- Best to place patient on a p.o. antibiotic to decrease risk of sinusitis and Toxic Shock Syndrome (due to staph aureus)
- Advise avoiding straining, bending forward or removing packing early
- Most patients may be treated as outpatients but hospital admission and observation should be strongly considered when a posterior pack is used. SaO2 should be monitored as well.
- Admission may also be prudent for those with CAD, severe HTN or significant anemia. Give supplemental oxygen via humidified face tent.

Epistaxis (cont.)

Acute Management

- ABC , 2 large bore IV Lines,
- cross match and transfuse if needed,
- PRBCs , FFP, PLTs. cyoprecipitate

Self Management

- Pinching the nose
- Bending the head over
- Waiting 5 mins
- Do not blow

- Preventive Measures

- Keep allergic rhinitis under control. Use saline nasal spray frequently to cleanse and moisturize the nose. Use saline-based gel intranasally for mucosal dryness
- Avoid forceful nose blowing
- Avoid digital manipulation of the nose with fingers or other objects
- Consider using a humidifier in the bedroom
- Keep vasoconstricting spray at home to use only prn epistaxis

- Blood Loss Management

- Blood loss Estimate
 - Vital signs*
 - Blood work up**
- Blood volume Expansion
- Blood Transportation
 - Blood Component

*hypotension start after 10% loss of blood volumes

**hematocrit is a good indicator of blood loss

- Other Treatments for Refractory Epistaxis

- Greater palatine foramen block
- Septoplasty
- Endoscopic cauterization
- Selective embolization by interventional radiologist
- Internal maxillary artery ligation (sometimes bilateral)
- Transantral sphenopalatine artery ligation
- Intraoral ligation of the maxillary artery
- Anterior and posterior ethmoid artery ligation
- External carotid artery ligation

Diseases of nasal septum

- The nasal septum is made up of bone and cartilage. **blood vessels between septum and cartilage.**
- It can be deviated, perforated, or collapsed.
- **collagen is a-vascular and obtains nutrition by diffusion. If there was hematoma this would lead to necrosis.**

❖ **The Nasal Septum Development** not mentioned by female doctor

1- Cartilaginous Septum

- Septal (quadrilateral) cartilage
- The vomeronasal cartilages
- Medial crura of the alar (lower lateral) cartilages

2- The Membranous Septum (Mobile Septum)

- Anterior to the end of the septal cartilage
- It is formed by skin and subcutaneous tissue of the nasal columella.
- The nose is lined by pseudostratified columnar epithelium except the anterior 1cm which is the membranous septum is lined by squamous epithelium.
- This membranous septum is lined by skin and hair so it will have skin disease rather than mucosal disease such as :hair follicles inflammation.

3- Bony septum:

- Composed of two major elements:
 - The Vomer: Develops from connective tissue membrane on each side of the septal cartilage. The intervening cartilage absorbed completed by mid adulthood.
 - The Perpendicular plate of the Ethmoid (Mesoethmoid): Ossification completed by 17th year of age. Replacement of cartilaginous septum with thin bone. At the nasal roof it articulates with the cribriform plate and extends as the crista galli.

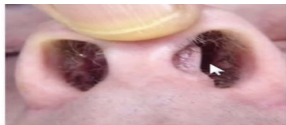
Diseases of nasal septum (cont.)

Inequality of Growth:

- Creating septal spur → Elevations and ridge like protuberances

1- Deviated nasal septum

- Approximately 80 % of humans have DNS, any or all parts of the septum except for the **posterior free border at the choanae**. A common area of deflection is along the **articulation between the vomer and the perpendicular plate of the ethmoid**.
- DNS to one side or S shape **example: anterior deviated to the left & posterior deviated to the right** to both side. The nasal septum is rarely exactly in the midline, minor deviations are normal and cause no symptoms, **marked** deviation will cause nasal airway obstruction and may contribute to sinonasal pathology by obstructing the normal sinus drainage pathways.
- Septal deviation can be corrected by surgery, with excellent results.
- Causes: Most cases of deviated nasal septum (DNS) result from **trauma**, either recent or long forgotten, perhaps during birth or childhood. 'Buckling' in children may become more pronounced as the septum grows. **Maldevelopment** → Congenital (considered in etiology in addition to trauma). **Nasal surgery**, including cosmetic surgery, can cause septal deviation. Spurs, crests, dislocation of quadrangular septal cartilage, buckling.



deviated septum

compensatory hypertrophy of turbinate to prevent entry of too much air compared to affected side. that's why the might complain of bilateral obstruction. 27



spur because only a part of septum is deviated

Diseases of nasal septum (cont.)

→ Signs & Symptoms:

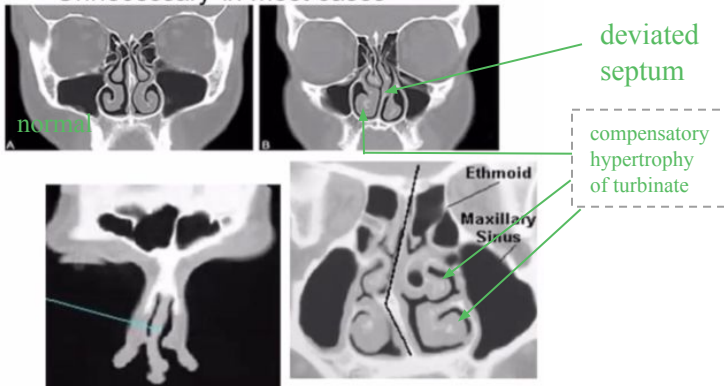
- Nasal obstruction (most important sx) may be unilateral or bilateral
- External deformity.
- Crusting, epistaxis (due to dryness caused by air hitting the mucosa directly) (a sharp spur can be a focus for epistaxis)
- Recurrent sinus infection due to impairment of sinus ventilation, the middle turbinate on the concave side of the septum may hypertrophy and interfere with sinus ventilation.
- Anterior can cause facial pain but this is rare.
- Otitis media: DNS may impair the ability to equalize middle-ear pressure
- Septal deviations are often found in patients with allergic rhinitis.

→ Diagnosis:

- The diagnosis is mostly clinical in deviated septum: elevations and ridge like protuberances, maxillary crest is groove for septum to set sometimes we find this groove projecting little pit as result of fault growth. As well as by endoscopy or speculum.
- Radiology is unnecessary in most cases only done when patient is suspected to have sinusitis
- NSD is a cause of sinusitis.
- As a compensation the turbinate hypertrophies

Radiology

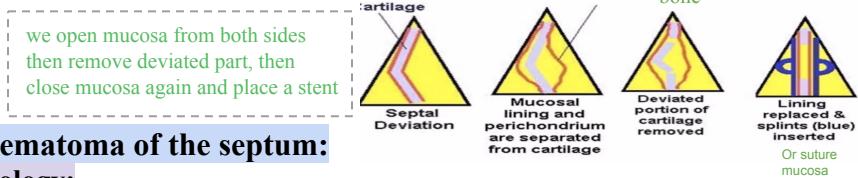
- Unnecessary in most cases



Diseases of nasal septum (cont.)

→ Treatment:

- If symptoms are minimal (asymptomatic) and there is only a minor degree of deviation, no treatment is needed.
- Submucous resection: obstructing cartilaginous and bony portion. Removal of deviated PART
- Septoplasty **if symptomatic**



2- Hematoma of the septum:

→ Etiology:

- Direct trauma. broken nose
- Operative trauma. “Septoplasty” surgery. we put stent to prevent it
- Blood dyscrasias. “bleeding disorders”/ on anticoagulant

→ Clinical features:

- **Obstruction.** main complaint. unilateral or bilateral
- Bleeding.
- Lacerations.
- Septal swelling

Unlike the turbinate the hematoma is: 1. red 2. soft 3. fluctuant 4. septal origin

→ Complications:

if you leave it it might cause abscess leading to destruction of cartilage and eventually perforation and nasal deformity

- **Cartilage necrosis, causing saddle nose deformity.**
- **Septal abscess.**
- **Cavernous sinus thrombosis**
- **Permanent thickening of the septum.**

→ Treatment:

- Immediate Incision and drainage.
- Systemic Antibiotics. As a prophylactic (don't forget dangerous triangle)



Diseases of nasal septum (cont.)

3- Perforation of the septum Imp to know diseases that cause septal perforation and those that cause bone perforation

→ Etiology:

- Nasal surgery.
- Trauma including repeated nose-picking.
- Infection
- Drugs **especially heroin because its a strong vasoconstrictor**
- (Chronic inflammation, e.g. nasal granulomatosis such as TB and sarcoidosis, syphilis. **it might also affect cartilage or bone**) **EXAM Q**
- Inhalation of fumes, e.g. chrome salts.
- Cocaine.
- Carcinoma.

→ Clinical features:

Clinical features depend on the size and the site of the perforation. A perforation is readily seen and often has unhealthy edges covered with large crusts.

- Asymptomatic
- Crusting **because ciliary movement is responsible for moving crust to nasopharynx so in case of perforation it will be interrupted so crust will collect at edge of perforation.** due to turbulence of air
- Epistaxis
- Whistling, “the smaller the size of the perforation the more the whistling”
“And the bigger the perforation → the more obstruction → due to air instead of going back to the nasopharynx there’s going to be turbulence “

→ Treatment:

- No treatment, in asymptomatic patients
- Nasal wash
- Surgical closure by silastic button or consider sliding or rotating mucoperichondrial flaps with or without a fascial graft; contraindicated for large perforations (approximately >2 cm of vertical height)

→ Diagnosis:

- Anterior rhinoscopy
- Biopsy of granulation tissue or abnormal mucosa

Diseases of nasal septum (cont.)

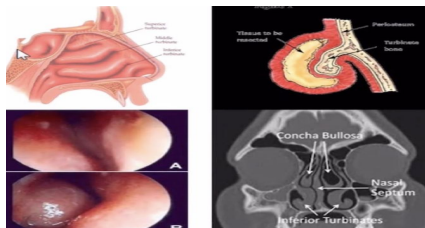
4- Turbinate Hypertrophy

Chronic Rhinitis leading to turbinate Hypertrophy **specially inferior**.
Common in both children and adults

→ causes:

- Infectious or non infectious
- Compensation
- Dysfunctional
- Allergic or non allergic

*NSD and tumor



→ manifestations

- Nasal obstruction
- mouth breathing

treatment depends on symptoms: if patient complains of obstruction.. then treat. If no sx even if it's size is big just leave it

Medical management 1st treat the cause if allergy treat it. we usually start by medical.

Surgical management just know there are multiple surgeries & the goal is to reduce turbinate size. do surgery if medical failed.

- Antihistamine
- Decongestant
- Topical nasal steroid, nasal saline, sinus rinses
- Antibiotic if sinusitis
- Immunotherapy if allergic

- Cold steel turbinectomy, turbinoplasty
- Lateralization/outfracture of inferior turbinate
- Diathermy (electrocautery)
- Laser
- Cryosurgery
- Powered microdebrider
- Radiofrequency ablation
- Coblation

-In surgery we don't do total turbinectomy we just remove part of it (partial turbinectomy), because it's important for protection of the nose. if you removed the whole turbinate, it might lead to atrophic rhinitis (crusting, bad smell of nose, obstructions, dryness..)

→ turbinate reduction goals:

- Mucosal preservation
- Controlled reduction
- Submucous scarring to reduce the erectile nature of the mucosa
- Bony reduction when necessary
- Minimal complications

Emergencies in nasal obstruction 437A

Diagnosis	Emergency	Complications
Septum Hematoma	Elevation of mucosal Septal cartilage, necrosis, development perichondrium with cartilage devascularization	Septal cartilage necrosis, abscess, development of a saddle-nose deformity
Septum Abscess	Intracranial extension of infection	Septal cartilage necrosis, development of a saddle-nose deformity, cavernous sinus thrombosis, intracranial infection
Mucormycosis	Tissue destruction	Extension to brain or orbit

Nasal operations

❖ Septoplasty:

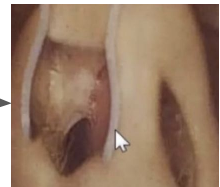
- Surgery involves elevating mucosal flaps from the septal cartilage and resecting part of the deviated cartilage straighten it and put it back in place (septoplasty, check the figure).
- Septal surgery should be undertaken with caution if at all in children as it may interfere with the growth of the mid-face.
- Nowadays we go in with certain techniques “we crush the deviated part with a specific tool for that” to repair the cartilage and put it back in place and also put splint inside “removable after 5 days”)

→ Indications of septoplasty:

- Nasal obstruction (deviated nasal septum)
- Epistaxis, chronic sinusitis (when septum is obstructing)
- Access for transseptal sphenoidotomy
- Headache from impacted spur
- Septal neoplasia(rare)

→ Complications of Septoplasty:

- Septal hematoma and abscess → due to infection
- Septal perforation
- Saddle nose deformity (over resecting cartilage anteriorly)
- Synechia (Adhesions) **between septum and lateral side of the nose** → will lead to obstruction.
- Cribriform plate fracture
- Anosmia
- Bleeding



Nasal operations

◆ Functional Endoscopic Sinus Surgery (FESS)

→ Indications for FESS:

- Chronic sinusitis
- Complicated sinusitis
- recurrent acute sinusitis, Failed medical management of acute sinusitis, fungal sinusitis, Obstructive nasal polyposis, Sinus mucoceles, Remove foreign bodies, Tumor excision, Transsphenoidal hypophysectomy, Orbital decompression, Dacryocystorhinostomy, Septoplasty, Orbital nerve decompression, Grave's ophthalmopathy, Choanal atresia repair, CSF leak repair, Control epistaxis, Orbital nerve decompression, Turbinectomy.

→ FESS goals:

-Eradication of disease. -Aeration. -Drainage. -Post op access.

→ The steps of FESS:

1. Medialized middle turbinate
2. Excise uncinat process
3. Anterior then posterior ethmoidectomies
4. Sphenoidotomy
5. Frontal recess sinusectomy
6. Create maxillary anrostomy

→ FESS Landmarks (CLOSE):

- 1-Cribriform plate
- 2- Lamina papyracea
- 3-Orbit
- 4- Sphenoid
- 5-Ethmoid
 - Complete extirpation of all the disease
 - Permanent drainage and ventilation of the affected sinuses
 - Postoperative access to the previously diseased areas.

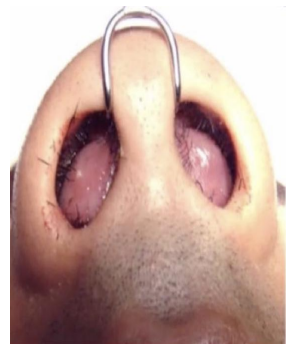
→ Postoperative Care:

- Sinus Packing.
- Oral Antibiotics for a minimum of 2 week.
- Aggressive nasal hygiene to prevent adhesions (saline irrigations).
- Nasal steroids.
- Nasal debridement at 1, 3, and 6 weeks.

Questions from Dr's slides:

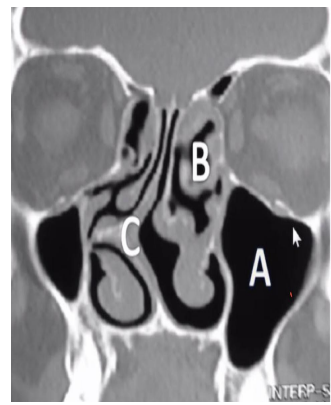
Q1. A 25 year old man post RTA with fever and nasal obstruction.

- What is your diagnosis? **Bilateral Septal Hematoma or abscess due the presence of FEVER.**
- What is your management? **Antibiotic and incision & drainage**



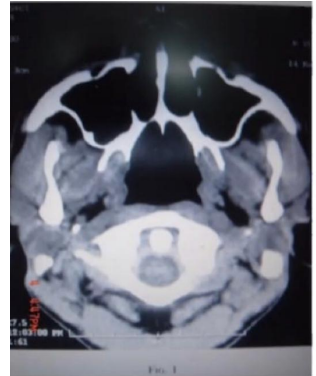
Q2.

- What is the Radiological study? **CT**
- What is A, B and C?
A is the maxillary sinus
B is the ethmoid
C is the deviated septum



Q3. This is a CT of a newborn who presented with respiratory distress.

- What is your diagnosis? **Bilateral choanal atresia**
- What is the management?
Initial: oropharyngeal airway
Definitive: perforation



Questions (thanks to 436)

1- A child presented with foul smell from the nose with discharge and obstruction what is the most common condition?

- A. Foreign body
- B. Polyp
- C. Allergic rhinitis
- D. Nasopharyngeal carcinoma

2- Young Patient came with unilateral nasal obstruction. In endoscope we found a pedunculated friable mass that bleeds with touch. What's the diagnosis?

- A. Nasopharyngeal cancer
- B. Inverted papilloma
- C. Allergic fungal sinusitis
- D. Chronic rhinitis

3- 20 year-old lady had aggressive inferior turbinectomies few years ago. Since then she is complaining of nasal obstruction, with foul nasal smell. What is the possible complication?

- A. Allergic rhinitis.
- B. Atrophic rhinitis.
- C. Chronic sinusitis.
- D. Nasal polyp

4- 30 year-old women complaining of headache increase on leaning forward during praying and mucopurulent post-nasal discharge. On examination, there was nasal discharge in both nasal fossae. What is the investigation required to reach the diagnosis?

- A. Skin allergy test.
- B. CT sinuses.
- C. Plain x-ray to the nasal bone.
- D. Full blood count.

5- A 20 years old patient was on antibiotics for acute frontal sinusitis. He developed diplopia, decreased vision in the left eye and pain behind the left eye. What is the diagnosis?

- A. Brain abscess
- B. Cavernous sinus thrombosis
- C. Preseptal cellulitis

answers:A/B/B/B/B

Questions (cont.)

6- 78 year-old patient noticed right sided nasal obstruction associated with a bloody discharge that had developed over the last month. What is the most likely diagnosis?

- A. Adenoidal hypertrophy.
- B. Allergic rhinitis.
- C. Carcinoma paranasal sinuses.
- D. Nasal polyposis.

7- A New born child had cyanosis and difficulty breathing immediately after delivery. The cyanosis improves with crying. Which of the following is the most likely diagnosis?

- A. Enlarged Adenoid
- B. Laryngomalacia
- C. Laryngeal web
- D. Bilateral choanal atresia

8- A 45 years old patient presented with progressive bilateral nasal obstruction. He was diagnosed as a case of bilateral nasal polyp. He underwent polypectomy and histological examination was required. What is the expected finding that confirms the clinical diagnosis?

- A. Benign neoplasm
- B. Granuloma
- C. Malignant neoplasm
- D. Oedematous mucosa

9- A 12-year-old girl is complaining of left unilateral nasal obstruction worse on expiration for 5 months. Examination of the nose showed a single pale grayish glistening pedicle mass in the posterior part of the left nasal cavity. A CT showed pacification of the left nasal cavity, maxillary sinus and the nasopharynx. What is the most likely diagnosis?

- A. Antrochoanal polyp
- B. Inferior turbinate enlargement
- C. Mucocele
- D. Juvenile angiofibroma

answers:C/D/A/A