Ryazan State Medical University

Department of surgical dentistry and maxillofacial surgery with a course of ENT diseases

Diseases of the nose, nasal cavity and paranasal sinuses

Practical guide
for students in the speciality of General medicine

Ryazan, 2021

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The practical guide provides information on diseases of the nose, nasal cavity and paranasal sinuses. This information is systematized and consistently presented. Knowledge of the classification, pathogenesis, clinical presentation and treatment will help future doctors to better diagnose and differentiate these ENT-diseases. The manual is intended for 4th year students studying in the specialty 31.05.01 «General medicine» and contribute to better mastering of theoretical material on otorhinolaryngology.
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Кафедра ХС и ЧЛХ с курсом ЛОР-болезней

Заболевания носа, полости носа и придаточных пазух носа

Практическое руководство
для обучающихся по специальности Лечебное дело

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З-125 Заболевания носа, полости носа и придаточных пазух носа: практическое руководство для обучающихся по специальности Лечебное дело / сост.: Т.И. Карасева; ФГБОУ ВО РязГМУ Минздрава России. – Рязань: ОТСиОП, 2021. – 83 с.

В практическом руководстве представлены сведения о болезнях носа, полости носа и придаточных пазух носа. Знание классификаций, патогенеза, клинической картины и методов лечения поможет будущим врачам лучше диагностировать и дифференцировать эти ЛОР-заболевания. Пособие предназначено для студентов 4 курса, обучающихся по специальности 31.05.01 «Лечебное дело» и способствует более качественному освоению теоретического и практического материала по оториноларингологии. Ил.: 59.

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Introduction

Diseases of the nose, nasal cavity and paranasal sinuses are the most common ENT pathologies. It is a significant health problem.

Rhinitis significantly reduces quality of life (QOL), interferes with both attendance and performance at school and work. The nose is the gateway to the respiratory tract and rhinitis is associated with symptoms arising from the sinuses, middle ear, the nasopharynx and lower airways. Occupational rhinitis often precedes the development of occupational asthma. Both allergic rhinitis (AR) and non AR are risk factors for the development of asthma. Rhinosinusitis can also be the presenting complaint of potentially severe systemic disorders such as Wegener's granulomatosis, sarcoidosis and Churg–Strauss syndrome. The ocular-orbital and intracranial complications of sinusitis constitute a diagnostic and therapeutic urgency that requires a correct multidisciplinary assumption.

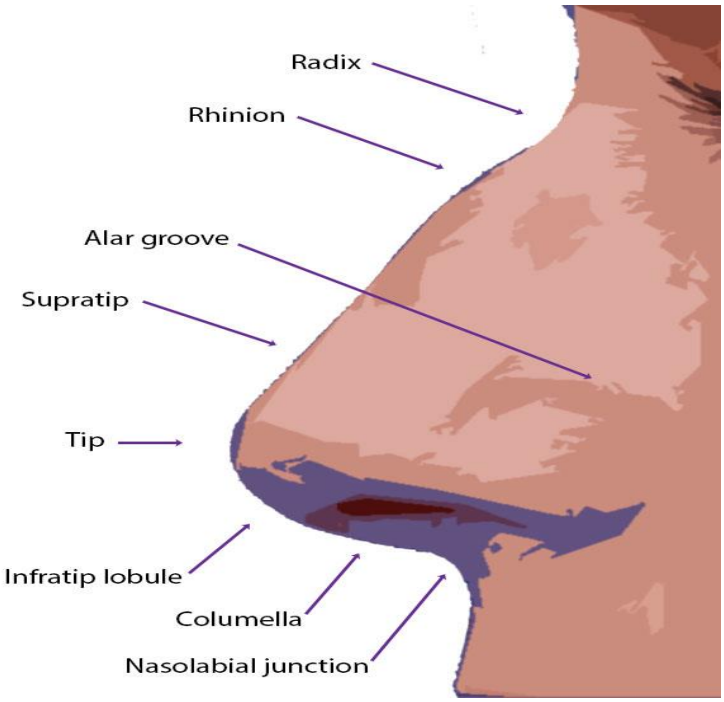
Epistaxis is a common clinical condition that can be challenging. Patients may present with intermittent blood streaking or life-threatening hemorrhage. The emergency physician must be ready to quickly evaluate and treat these patients, needs knowledge of the procedures to control epistaxis and when a referral to an otolaryngologist is indicated.

Injuries to the nose and paranasal sinuses often result in a combination of aesthetic and functional problems that may alter a patient's lifestyle. Patients experiencing trauma can be devastated by the alteration to their appearance with its concomitant psychosocial implications. Functional disturbances can affect breathing, olfaction, eating and chewing, and vision.

The aim of our textbook is to evaluate the clinical features, radiological finding, bacteriology, treatment of the diseases.

Nose anatomy

External Surface Landmarks



The nose is commonly divided into 3 areas: the upper, middle and lower thirds. These 3 areas can be corrrelated clinicially to the surface landmarks of nasion, rhinion and tip, respectively.

Pic. 1. Surface landmarks of the nose

Soft Tissue of the External Nose

The soft tissue anatomy of the nose can be simplied into 4 layers: skin, fat, fascia and muscle.

Skin & Fat

Upper 2/3 is thin.

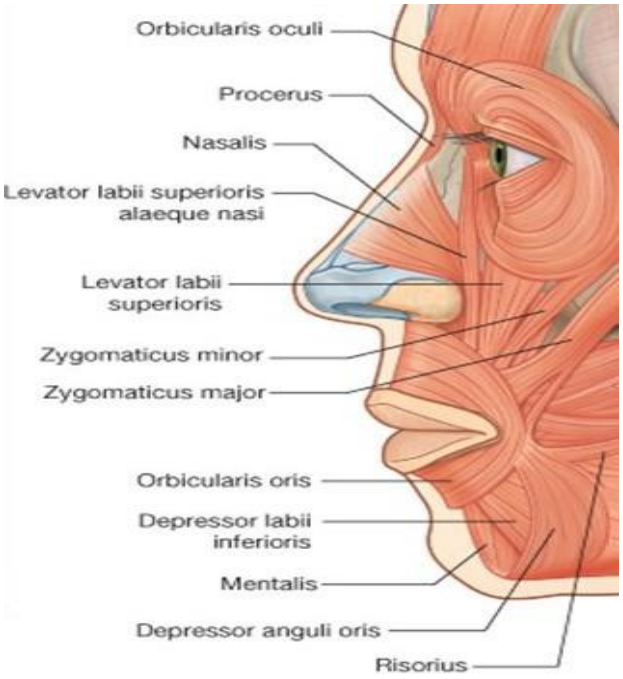
Lower 1/3 is less mobile, more sebaceous.

In some areas, there is a supeficial fatty and deep fatty layer.

Fascia surrounding the nose is a fibromuscular layer. This includes nasal subcutaneous muscular aponeurotic system (SMAS).

Muscles

The muscles of the face subdivide into four groups. These groups consist of the orbital group, the nasal group, the oral group, and others. The muscles correlating with the nasal group are the nasalis, procerus, and depressor septi. Another muscle, levator labii superioris alaeque nasi, is associated with the oral group but has functions associated with the nose. The facial nerve, CNVII, is the innervation for these muscles.



Pic. 2. Muscles of the face

Nasalis

• transverse part: Originates on the maxilla lateral to the nose and inserts on the dorsum of the nose. This part compresses the nasal aperture.

• alar part: Originates above the lateral incisor and inserts on the alar cartilage. The function of this part is to open the nostril by pulling the alar cartilage down and lateral.

Procerus

The procerus originates on the nasal bone and upper lateral cartilage while inserting on the skin overlying the glabella. The function of the procerus is to wrinkle skin over the bridge of the nose by pulling the medial angle of the eyebrows downward.

Depressor septi

The depressor septi originates on the maxilla above the central incisors and inserts on the anterior septum. This muscle's primary function is to draw the nose inferiorly.

Levator labii superioris alaeque nasi (LLSAN)

The LLSAN originates on the frontal process of the maxilla and inserts on the alar cartilage and upper lip. The function of this muscle is to open the nostril and elevate the upper lip.

Bone & Cartilage of the nose

The nasal cavity is an osseo-cartilaginous framework divided into 3 main vaults. The upper 1/3 is bone, the middle and lower thirds are cartilage.

Upper 1/3 - Bony Vault

The "Bony Vault" is a pair nasal bones surrounded by frontal, maxillary and ethmoid bones. It has a characteristic hour-glass figure and is generally thicker and more dense above the medial canthus.

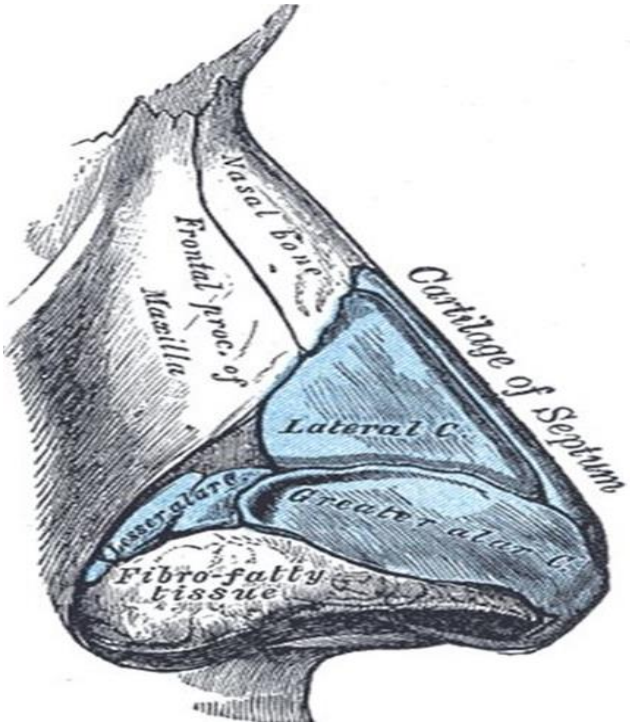
In relation to adjacent bones, the nasal bone interdigitates with:

• superior: Nasal process of the frontal bone
• lateral: Frontal process of the maxilla
• posterior: Perpendicular plate of the ethmoid
• medial: paired nasal bone

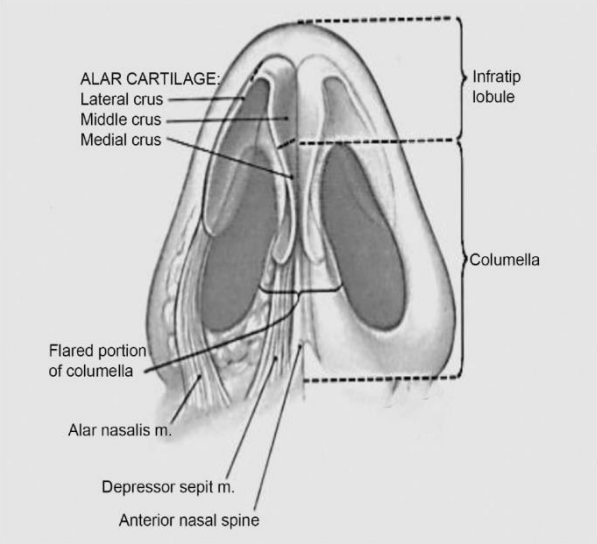
Middle 1/3 - Cartilaginous Vault

The middle 1/3 of the nasal cavity is a cartilaginous structure of:

• paired upper lateral cartilages (ULC) creates an internal nasal valve at the ULC's caudal edge and nasal septum. This is the narrowest part of the nasal airway
• dorsal septum
• soft tissue attachments.



Pic. 3. The Upper and Middle 1/3 of the Nose



Pic. 4. The Lower 1/3 of the Nose

Lower 1/3 - Cartilaginous Vault

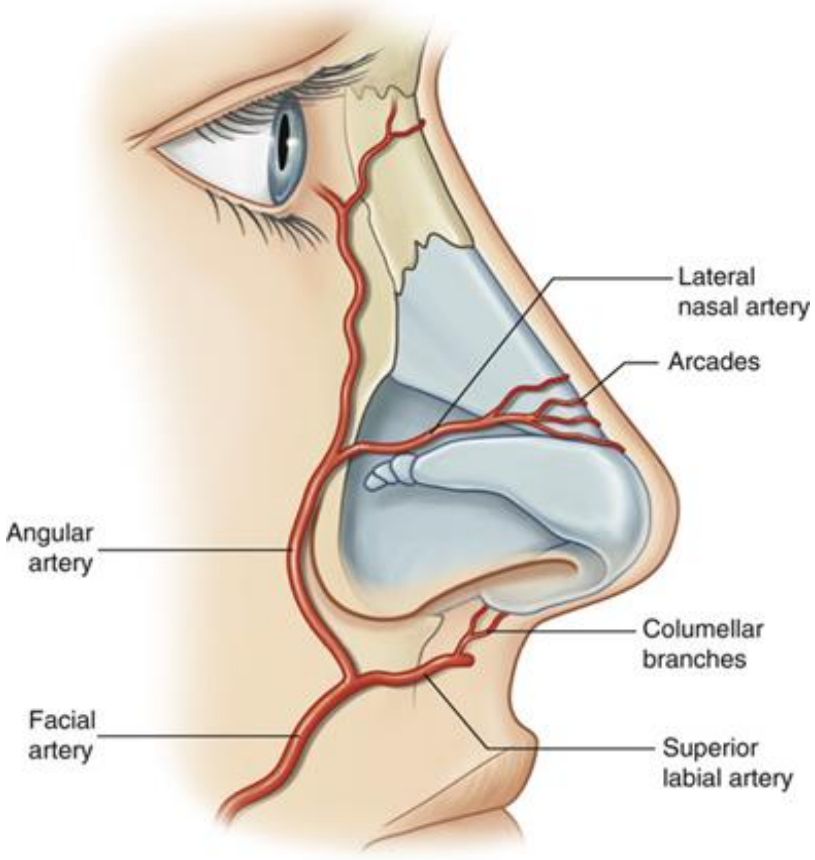
The lower vault is a cartilaginous structure composed of paired lower lateral cartilages (LLC), which strongly influences tip projection, rotation, and definition. The junction between the ULC and LLCs is called the scroll area.

It can be divided into 3 crus:

• medial: runs through the columella.

• middle: forms the nasal tip.

• lateral: passes superolateral from tip. Shares a common perichondrium with accessory cartilage.



Pic. 5. Blood supply to the nose

Blood supply to the nose

The primary blood supply of the nose is the facial and ophthalmic arteries.

Facial artery

The facial artery provides blood supply to the nasal tip two branches:

• columellar branches of the superior labial artery

• lateral nasal branch of the angular artery.

Ophthalmic artery

The opthalmic artery supplies the superior portion of the nasal envelope via:

• anterior ethmoidal artery.

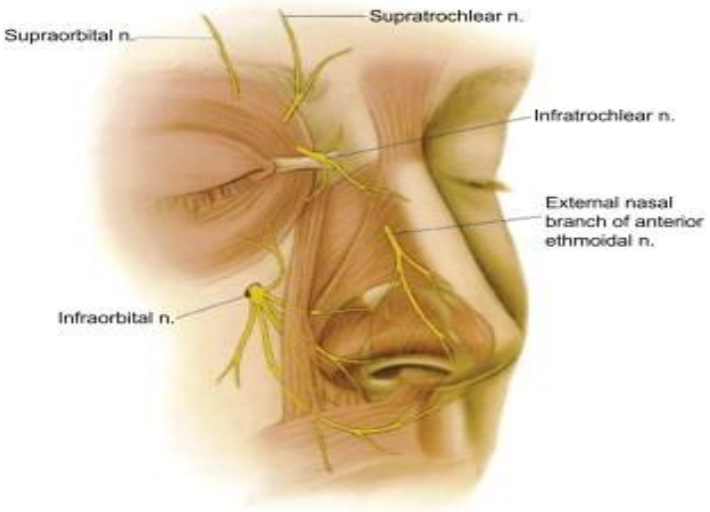
• dorsal nasal artery.

• external nasal artery (branch of ophthalmic artery).

Nerve sensation to the nose

External nasal skin is supplied by ophthalmic (V1) & maxillary (V2) branches of trigeminal (CN V) nerve.

Branches of the ophthalmic nerve



Infratrochlear and supratrochlear nerves supply radix and cranial part of dorsum.

External nasal branch of the anterior ethmoidal nerve supplies the caudal part of the dorsum and tip. This emerges between the nasal bones and upper lateral cartilages.

Pic. 6. Innervation to the nose

Branches of maxillary nerve

Infraorbital nerve supplies lateral nose and tip.

The nasal branch from the anterior superior alveolar nerve supplies the columella.

The external nasal branch of the anterior ethmoid innervates middle vault and nasal tip.

Internal nasal anatomy

Introduction

The role of the nasal cavity is to humidify and warm the inspired air. Also, as the air passes through, the nasal cavity removes minute airborne particles and other debris before the air reaches the lower airways. Columnar epithelium lines the nasal cavity. This type of epithelial lining also secretes mucus that coats the lining and helps with the mucociliary clearance of minute aerosolized particles that become trapped in the nasal mucosa. The nasal cavity also functions to facilitate drainage for the secretions from the adjacent paranasal sinuses. It also captures the odor bearing particles and transmits them to the olfactory recesses, that are in the superior portion of the nasal cavity, just medial to the superior turbinates. Air containing mucosal lined sinuses surround the nasal cavity, which includes the frontal, maxillary, sphenoid and ethmoid sinuses. These cavities directly communicate with the nasal cavity. The secretions from these sinuses drain into the nasal cavity via the thin-walled ostia. Like the nasal cavity, the wall lining of the sinuses also secretes mucus. The cilia on the surface sweep the mucus in a carpet like fashion and move them towards the nasal ostia. The hard palate lines the floor of the nasal cavity. The lateral walls are spiral shaped mucosal folds that overlie the turbinates and sinus ducts draining into the ostia. The spiral shape of the turbinates is designed to increase the surface area for the inspired air.

Structure and Function

The nasal cavity is the most cephalic part of the respiratory tract. It communicates with the external environment via the anterior apertures, nares, and the nasopharynx via the posterior apertures, choanae.

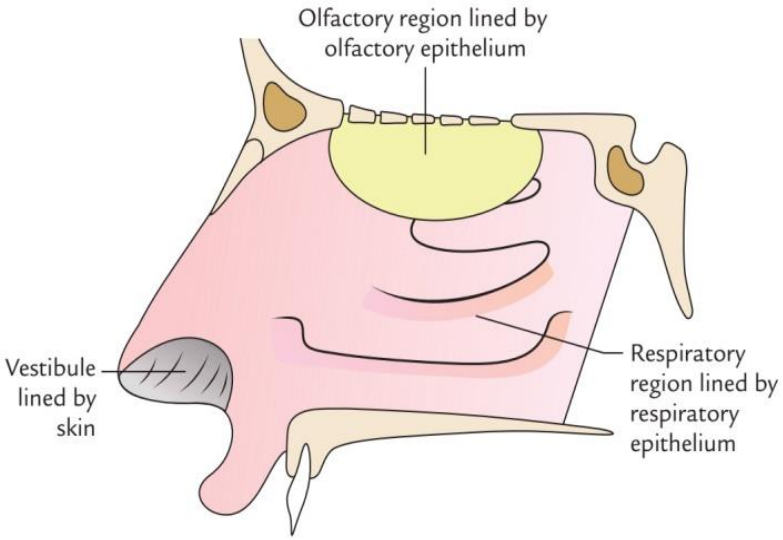
This cavity is divided into two separate cavities by the septum and kept patent by a bone and cartilaginous framework. Each cavity consists of a roof, floor, medial wall, and lateral wall. Within each cavity are three regions; nasal vestibule, respiratory region, and olfactory region.

Respiratory Region

The respiratory region functions to humidify, warm, filter, protect, and eliminate debris. Covered in respiratory epithelium and mucous cells, this is the most substantial part of the nasal cavity. As

air traverses through the nasal cavity, it warms to body temperature and reaches near one hundred percent humidity. The neurovascular supply of this region aids this. It regulates the nasal airflow by controlling the blood volume in the erectile tissue on the inferior turbinate and anterior septum. Under normal conditions, this tissue is continuously stimulated by sympathetic signals via the superior cervical ganglia to keep the nasal cavity uncongested.

Particles that get past the nasal vestibule then become trapped in the mucosa of the nasal cavity. When this occurs, the mucociliary system helps get rid of these particles. The ciliated pseudostratified columnar epithelium sweeps particles at a rate of one centimeter per minute into the nasopharynx for further expulsion.



The mucus of the nasal cavity forms a protective barrier to inhaled pathogens. The components of the mucus that actively protect the host are immunoglobulin A, lysozymes, and lactoferrin.

Pic. 7. Olfactory region

Olfactory Region

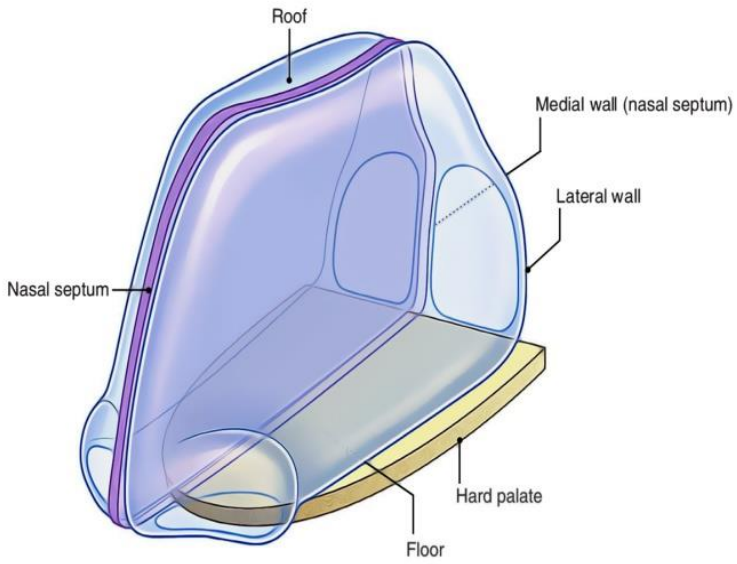
Olfaction requires orthonasal or retronasal airflow to transport odor-bearing particles up to the olfactory epithelium located at the apex of the nasal cavity. As odorants become trapped in the mucus, it binds to odorant binding proteins that concentrate and help solubilize the particles. The particles are then attached to olfactory receptors on cilia that transmit specific signals up through the cribriform plate to synapse with neurons of the olfactory bulb, which then sends signals through the olfactory nerve (CNI) into the secondary neurons for higher processing before entering the brain. A unique feature of the

olfactory receptors is that a single receptor cell can detect only one odorant type and cannot regenerate.

Nasal Vestibule

The nasal vestibule is the first area encountered as you move posteriorly through the anterior nares, also known as the nostrils or external nasal valve. The first half of the vestibule has a covering of keratinized stratified squamous epithelium that contains coarse hairs called vibrissae. These hairs filter inhaled particles. The covering of the second half of the vestibule is in respiratory epithelium, pseudostratified ciliated columnar epithelium.

• lateral: lateral crus of the lower lateral cartilage (LLC) and fibrofatty alar tissue
• medial: medial crus of the LLC and septal cartilage
• posterior: limen naris
• anterior: nasal spine of the frontal bone and nasal bone
• posterior: cribriform plate of the ethmoid and the body of the sphenoid



The roof of the nasal cavity

The mucosa of the roof of the nasal cavity contains perforations that communicate with the cribriform plate. Within these perforations are the olfactory axons.

Pic. 8. Nasal cavity

The floor of the nasal cavity

The floor of the nasal cavity is broader than that of the roof.

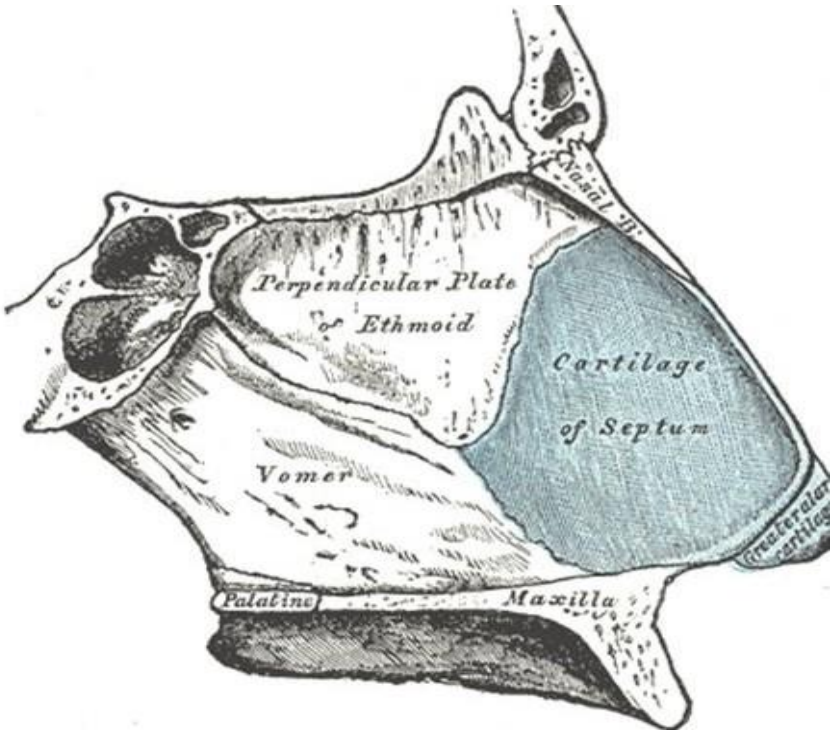
• anterior: the palatine process of the maxilla
• posterior: horizontal plate of the palatine bone

Incisive canal

This canal is located in the floor of the nasal cavity, posterior to the central incisor, and lateral to the nasal septum. This structure transmits the nasopalatine nerve into the oral cavity and the greater palatine artery into the nasal cavity.

Nasal septum

The nasal septum is a cartilaginous (cranial) and membranous (caudal) structure.



Pic. 9. Nasal septum

The nasal septum structure is composed of:

• anterior: quadrangular cartilage (It contains the Kiesselbach plexus)

Attachments:

Superior: nasal bone

Inferior: anterior nasal spine of the maxilla

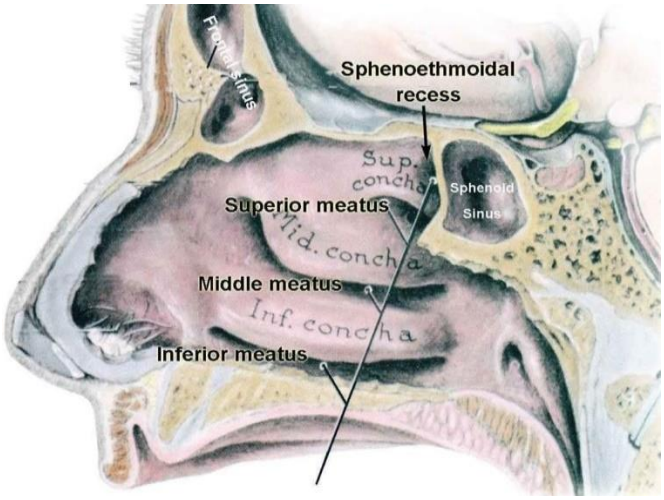
Posterior-Superior: perpendicular plate of the ethmoid

Posterior-Inferior: vomer and maxillary crest

• superior: perpendicular plate of ethmoid.

• inferior: vomer, nasal process of maxilla, perpendicular plate of palatine bone.

The nasal cavity's lateral wall has three medially projecting inferiorly curved bones called conchae. The middle and superior conchae are part of the ethmoid bone, whereas the inferior concha is a separate bone altogether. There is a normal variant called the supreme conchae. These conchae, when covered by mucosa, are termed turbinates. The turbinates augment the surface area of the nasal cavity to aid in its functions of humidifying, warming, and humidifying the air. The turbinates create four channels.



Three of these channels are termed meatuses, and the fourth is the sphenoethmoidal recess.

Pic. 10. Lateral wall of the nasal cavity

Bones of the lateral wall:
• ethmoid bone
• perpendicular plate of the palatine bone
• the medial plate of the pterygoid process of the sphenoid bone
• medial surface of the lacrimal and maxillary bones
• inferior concha

Sphenoethmoidal Recess: Located superior to the superior turbinate and inferior to the nasal cavity roof, which is the drainage site of the sphenoid sinus.

Meatuses

Superior Meatus: located inferior to the superior turbinate and superior to the middle turbinate; this is the drainage site of the posterior ethmoid sinus.

Middle Meatus: located inferior to the middle turbinate and superior to the inferior turbinate - there are several structures within this meatus. This is the drainage site of the frontal, anterior ethmoid, and maxillary sinuses.

Inferior Meatus: Located inferior to the inferior turbinate and superior to the floor of the nasal cavity. The nasolacrimal duct drains tears from the lacrimal sac at the medial aspect of the eye into the anterior portion of this meatus via Hasner's valve.

Limen naris: The limen naris is a mucosal ridge that signifies the posterior boundary of the nasal vestibule and the anterior boundary of the nasal cavity proper.

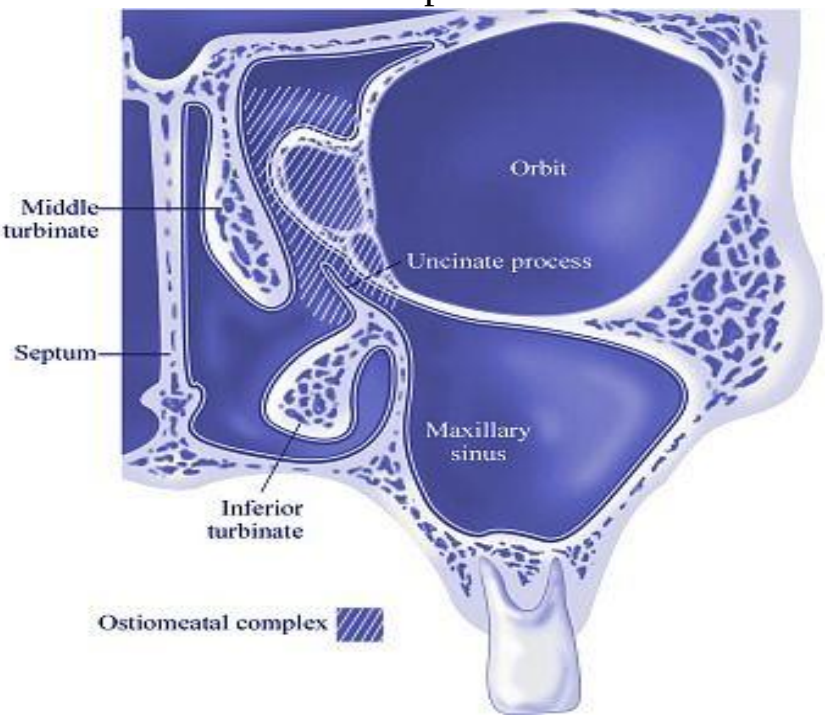
Agger nasi cells: These cells are the most anterior portion of the anterior ethmoid air cells. They are located anterior and superior to the basal lamella, most anterior attachment to the lateral wall, of the middle turbinate to create the anterior aspect of the frontal recess.

Frontal recess: Located between the posterior wall of the agger nasi cells and the middle turbinate.

Uncinate process of the ethmoid: This is a thin crescent-shaped bone that is part of the ethmoid bone. It is attached to the lacrimal bone anteriorly, the inferior turbinate inferiorly and superiorly to the lamina papyracea. This structure protects the sinuses of the infundibulum from inhaled foreign particles.

Lamina Papyracea: This thin bone is the separation between the orbit and the ethmoid air cells.

Ethmoid infundibulum: This is a pyramidal shaped channel located at the anterior portion of the semilunar hiatus that drains the maxillary, anterior ethmoid, and frontal sinuses.



Semilunar hiatus: Located between the uncinate process anteriorly and the ethmoid bulla posteriorly, this is a space that empties the ethmoid infundibulum.

Pic. 11. Ostiomeatal complex

Ethmoid bulla: Located just anterior to the semilunar hiatus and superior to the ethmoid infundibulum, which is where the middle ethmoidal air cells open into the nasal cavity.

Ostiomeatal complex (OMC): This is an area located lateral to the middle turbinate that houses the ostia of the lateral wall sinuses; frontal, maxillary, and anterior/middle ethmoid sinuses.

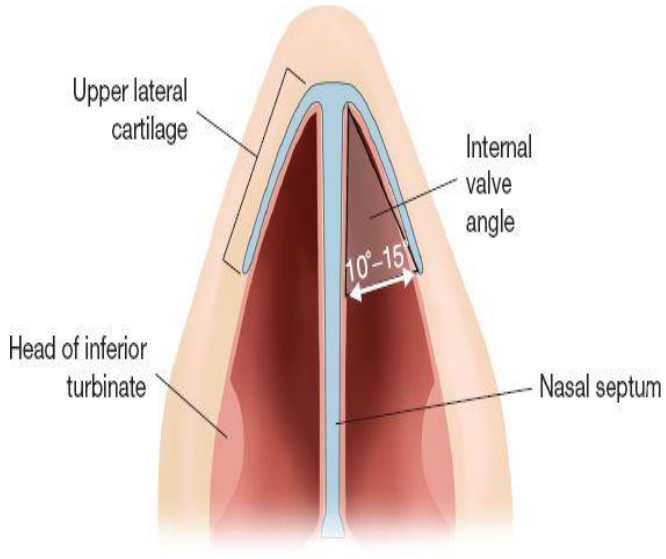
Sphenopalatine Foramen: This foramen connects the nasal cavity to the pterygopalatine fossa and is posterior to the middle turbinate in the posterior portion of the superior meatus. The significant content of this foramen is:

• sphenopalatine artery of the maxillary artery
• nasopalatine branch of the maxillary nerve of the trigeminal nerve (CNV2)
• posterior superior lateral nasal nerves of CNV2

Choanae

The choanae are also known as posterior nasal apertures. It is the posterior boundary of the nasal cavity proper. It opens into the nasopharynx.

• superior: the body of the sphenoid bone
• inferior: horizontal plate of the palatine bone
• lateral: the medial pterygoid process of the sphenoid bone
• medial: vomer



Pic. 12. Internal nasal valve (INV)

The INV is the narrowest portion of the nasal cavity and constitutes the area of highest resistance to airflow, which causes an increase in the acceleration of airflow. Without proper support, this

increased airflow causes a decrease in intraluminal pressure, which ultimately causes the INV to collapse; this is Bernoulli's principle of flow. The average cross-sectional area of the INV in adults is around 0.73 square centimeters. At the apex of the valve the ULC and, the nasal septum come together at an angle of 10 to 15 degrees.

- superior: upper lateral cartilage (ULC/caudal edge)
- inferior: nasal floor or hard palate
- lateral: the anterior portion of the inferior turbinate
- medial: nasal septum

Blood Supply and Lymphatics

Arterial supply

The nasal cavity has an abundant supply of vasculature to aid in functions of warming and humidifying inhaled air. It allows the mucosa to enlarge and shrink, under the influence of sympathetic innervation.

The arterial supply to the nose and nasal cavity originates from the internal and external carotid arteries.

Internal carotid artery (ICA)

The primary branch off of the ICA that supplies the nasal cavity is the ophthalmic artery. Coming off of the ophthalmic artery are the anterior and posterior ethmoid arteries, as well as the dorsal nasal artery.

The anterior ethmoid artery supplies the lateral nasal wall and the nasal septum. The posterior ethmoid artery supplies the superior turbinate and the nasal septum. The dorsal nasal artery supplies the dorsal aspect of the external nose.

External carotid artery (ECA)

The ECA gives rise to the maxillary artery and the facial artery. These two significant arteries then branch into smaller vessels.

Maxillary artery

The maxillary branches into the descending palatine artery that then travels through the pterygopalatine fossa down the palatine canal and then branches into the greater and lesser palatine arteries. The greater palatine artery then enters the greater palatine foramen on the posterior aspect of the palate before traversing the palate anteriorly to enter the nasal cavity via the incisive canal. It supplies the septum and the floor of the nasal cavity.

Like the descending palatine artery, the sphenopalatine artery is a branch of the maxillary artery. It branches off of the maxillary artery near the pterygopalatine fossa where it then enters the lateral wall of the nasal cavity through the sphenopalatine foramen, located just posterior to the medial turbinate.

The sphenopalatine artery then branches into the posterior lateral nasal branches and the posterior septal branch. The posterior lateral branches supply the middle and inferior turbinates, while the posterior septal branch supplies the posterior septum.

Facial artery

The facial artery gives rise to the superior labial artery, the lateral nasal artery, and the angular artery.

The superior labial artery gives off an alar branch and a septal branch that supply the same structures as their name. The lateral nasal artery supplies the alar cartilage on the external nose and also supplies the nasal vestibule. The angular artery supplies the external nasal tip, dorsum, and lateral wall.

Kiesselbach's plexus (Little's Area)

Kiesselbach's plexus is a vascular anastomosis between the anterior ethmoid artery, superior labial artery, greater palatine artery, and the terminal branch of the posterior septal branch of the sphenopalatine artery. This vascular plexus is located in the anterior nasal septum and is the most common site of epistaxis.

Woodruff's plexus

Woodruff's plexus is a vascular anastomosis between the sphenopalatine artery and the ascending pharyngeal artery. Located on the lateral wall of the nasal cavity in the area posterior to the middle and inferior turbinates.

Venous drainage

The names of the veins that drain the nose and nasal cavity follow that of the arteries with which they pair. The maxillary branches drain either into the cavernous sinus or the pterygoid plexus located in the infratemporal fossa. The veins of the anterior nasal cavity drain into the facial vein. Of note, infections

located between the oral commissure and nasal bridge, have the potential to become intracranial infections.

Lymphatics

The anterior nasal cavity drains anteriorly to the face that then makes its way to the submandibular lymph nodes. The lymphatics of the posterior nasal cavity and paranasal sinuses drain into the upper cervical lymph nodes and retropharyngeal lymph nodes.

Nerves:

Olfactory Nerve (CNI)

The olfactory nerve transmits signals from the nasal cavity to the brain to give the sense of olfaction.

The olfactory epithelium is in the superior portion of the nasal cavity. Within this epithelium are sensory cilia that project up through the cribriform plate to the olfactory bulb. From the olfactory bulb, signals are sent through the olfactory nerve proper to a network of secondary neurons for processing before ending up in the brain.

Trigeminal Nerve (CNV)

The trigeminal nerve is the sensory innervation to the external and internal nose. The branches are the ophthalmic (V1), maxillary (V2), and mandibular (V3). Sympathetic and parasympathetic fibers run with these branches to supply their target tissues. The ophthalmic and maxillary branches innervate the nose and nasal cavity.

Ophthalmic Branches (V1): As the ophthalmic nerve begins to branch, it gives off a nasociliary branch, which then provides the anterior and posterior ethmoid nerves. The anterior ethmoid gives off an external branch that supplies the nasal tip, an internal branch that supplies the anterosuperior nasal cavity and a septal branch that supplies the anterior superior nasal septum.

The posterior ethmoid supplies the posterosuperior nasal cavity. Two other branches of the ophthalmic branch of the trigeminal nerve are the supratrochlear and infratrochlear nerves that supply the nasal dorsum.

Maxillary Branches (V2): The maxillary branches of the trigeminal nerve that innervate the nose and nasal cavity branch in or near the pterygopalatine fossa then enter the nasal cavity. The only external nasal branch is the infraorbital nerve, which supplies the malar and lateral nose.

The nasopalatine nerve traverses the nasal septum from posterior to anterior in a downward projection to enter the incisive canal. It supplies the posterior and inferior nasal septum as well as mucosa just

posterior to the incisors. The greater palatine nerve follows the greater palatine artery down the palatine canal, giving off posterior inferior lateral nasal nerves that supply the posterior lateral wall of the nasal cavity. Three other nerves come off the maxillary branch (V2).

Two of these are the posterior superior lateral nasal nerve and posterior superior medial nasal nerve, both of which pass through the sphenopalatine foramen to supply the lateral and medial walls of the nasal cavity, respectively. The superior alveolar nerve is the last branch of V2, and it supplies the anterior septum and the area near the nasal vestibule.

Anatomy of the paranasal sinuses

Maxillary Sinus (Antrum of Higmore)

The maxillary sinus is a pneumatic space. It is the largest bilateral air sinus located in the body of the maxilla and opens in the middle nasal meatus of the nasal cavity with single or multiple openings. It is an air cavity with a quadrangular pyramidal shape with various walls:

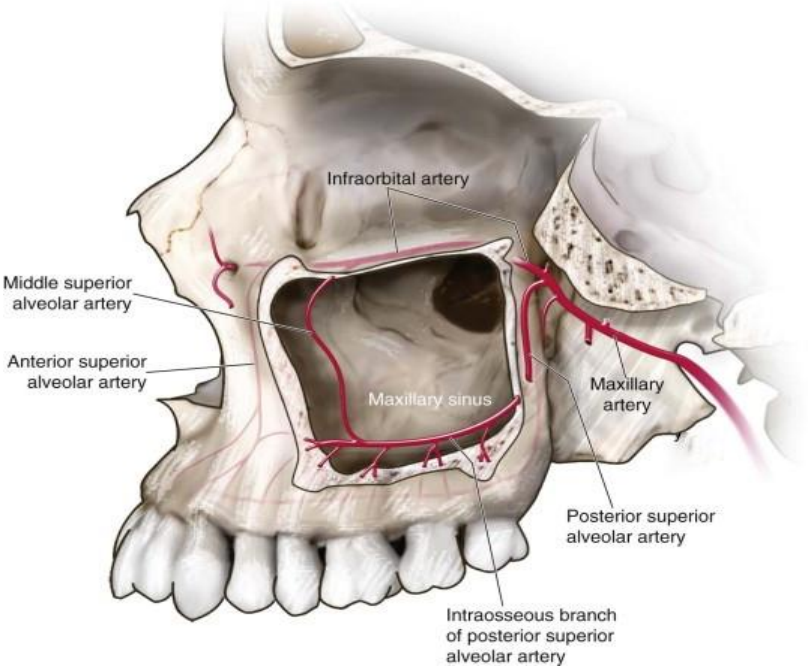
• medial wall facing the nasal cavity
• posterior wall facing the maxillary tuberosity
• mesio-vestibular wall for the presence of the canine fossae (it is only 2mm in thickness, though this canine fossa area that maxillary antrum is entered during Caldwell Luc surgery)

Boundaries of canine fossa:

• inferior: bounded by the alveolar ridge
• laterally: bounded by the canine eminence which is caused by the canine tooth.
• superior: infraorbital foramen
• medial: pyriform aperture (this does not contain bone, but is lined by middle meatus mucosa, a layer of connective tissue and the sinus mucosa)
• upper wall which is the orbit floor (this wall is very fragile and any disease process involving the maxilla is likely to affect the orbit through this wall, this wall is further thinned out where the infra orbital canal is present).

• lower wall that is next to the alveolar process and which is the bottom of the maxillary sinus itself (the roots of the first and second molar reach up to the floor of the maxillary sinus; in children the floor lies at the same level as that of the nasal cavity, in adults it lies 5 – 10 mm below the nasal cavity, it is just separated from the floor of the sinus by a thin lamella of bone; this lamella may be dehiscent commonly, dental infections involving the 1st and 2nd molars may involve the maxillary sinus through this thin lamella of bone).

The maxillary sinus communicates with the homolateral nasal fossa by means of a natural ostium located antero-superiorly on the medial surface, which drains into the middle meatus.



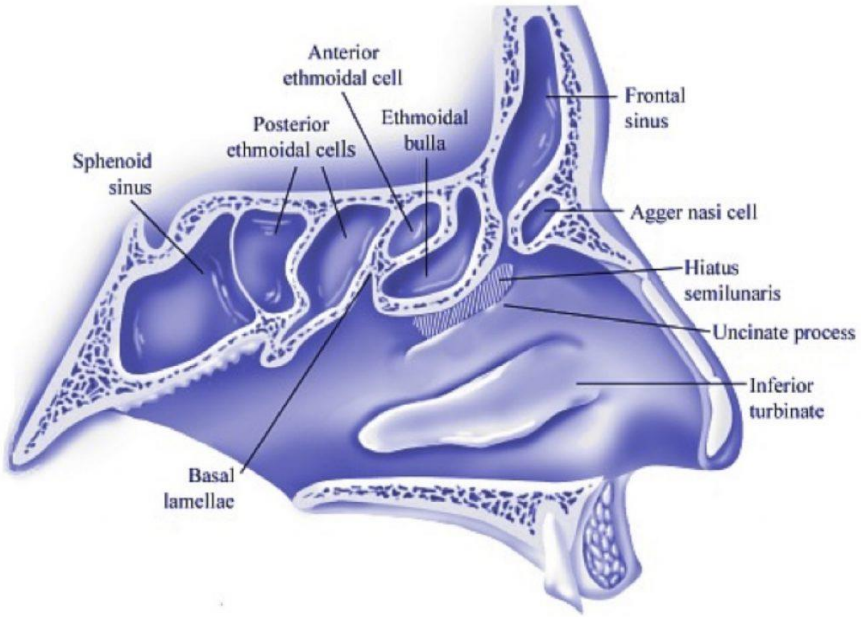
Pic. 13. Blood supply of the maxillary sinus

The blood supply of the maxillary sinus comes from the maxillary artery. Posterior superior alveolar artery (PSAA) and infraorbital artery are the branches of maxillary artery that supply lateral sinus wall and overlying membrane. Anatomically, anastomosis between the PSAA and anterior superior alveolar artery from infraorbital artery is always found at the lateral antral wall.

Innervation

The maxillary sinus receives general sensation innervation from the infraorbital and anterior, middle, and posterior superior alveolar branches of the maxillary nerve (V2). The ostium of the maxilla is innervated by the greater palatine nerve while the infundibulum is

innervated by the anterior ethmoidal branch of the ophthalmic nerve (V1). Parasympathetic secretomotor fibers originate from the nervus intermedius of the facial nerve, synapsing in the pterygopalatine ganglion and proceeding to the sinus mucosa via the trigeminal sensory branches.



Pic. 14. Ethmoid sinus

Ethmoid sinus:

The ethmoid sinus is referred to as ethmoidal labyrinth because of the complexity of anatomy. A collection of air cells (3-18 in number) separated by bony septa within each side of the lateral mass, or labyrinth, of the ethmoid bone.

They are separated into anterior and posterior groups by the basal lamella, the lateral attachment of the middle turbinate to the lamina papyracea. Historically the ethmoid sinuses were subdivided into 3 groups of air cells: the anterior, middle and posterior ethmoidal air cells. The middle group are now incorporated into the anterior group. Some of the ethmoidal air cells have been given specific names, because of their importance in surgical procedures or involvement in head and neck pathologies: frontal recess cells, including the agger nasi cells, ethmoid bulla, Haller cells, Onodi cells.

Boundaries:

Lateral wall: is formed by the orbital plate of the ethmoid otherwise known as the lamina papyracea.

This is a thin lamina of bone separating the orbit from the ethmoidal air cells. This wall could be dehiscent (normal variant).

Infections involving the ethmoid air cells may spread to the orbit through this wall.

Roof: is formed by the frontal bone anteriorly, this area is known as fovea ethmoidalis, and by the face of sphenoid and orbital process of palatine bone posteriorly. The frontal bone component of the ethmoidal roof is very thick. The transition of this thick fovea to the thin portion of roof of ethmoid medially is very weak. This is infact the weakest portion of this area and is prone to injuries during surgery leading on to csf leak.

The anterior ethmoidal air cells drain to the hiatus semilunaris and middle meatus via the ethmoid bulla, which forms parts of the ostiomeatal complex. The posterior ethmoidal air cells drain to the superior meatus via the sphenoethmoidal recess.

Arterial supply

From the ophthalmic branch of the internal carotid artery, the supraorbital, anterior and posterior ethmoidal arteries supply the ethmoid air cells with the sphenopalatine artery (a branch of the maxillary artery) also contributing. Thus, the ethmoid air cells are supplied by branches of both the internal and external carotid arteries.

Lymphatic drainage

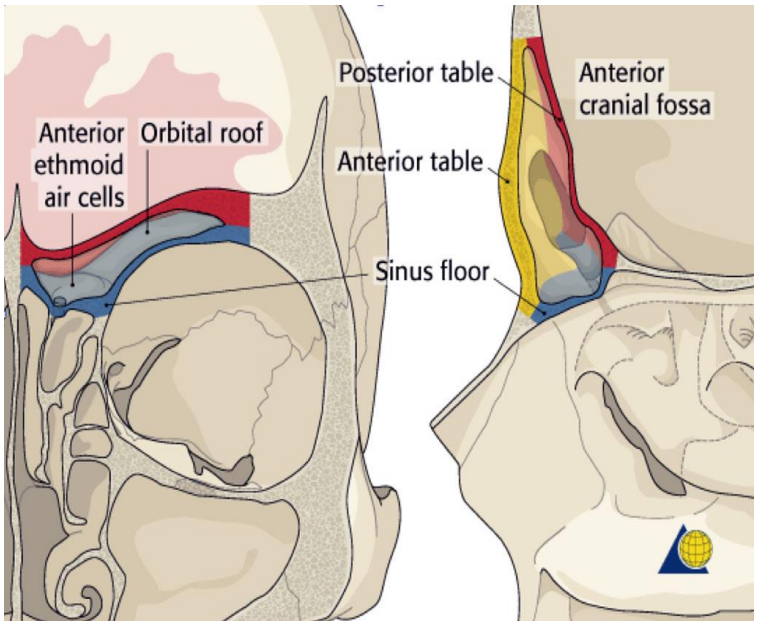
Lymph from the ethmoid air cells drains to the submandibular and retropharyngeal group of nodes.

Innervation

The posterior ethmoidal air cells, along with the sphenoid sinus, are supplied by the posterior ethmoidal nerve, whereas the anterior ethmoidal nerve supplies the anterior ethmoidal air cells. Both these nerves are extraconal branches of the nasociliary nerve, a branch of the ophthalmic division of the trigeminal nerve.

Frontal sinus

There are two sinuses extending in the squamous part of the frontal bone. They are separated by bony septum because each sinus (right and left) develops independently; they are expected to be asymmetrically pneumatized. The larger sinus may pass across the midline and overlap the other.



Pic. 15. Frontal sinus

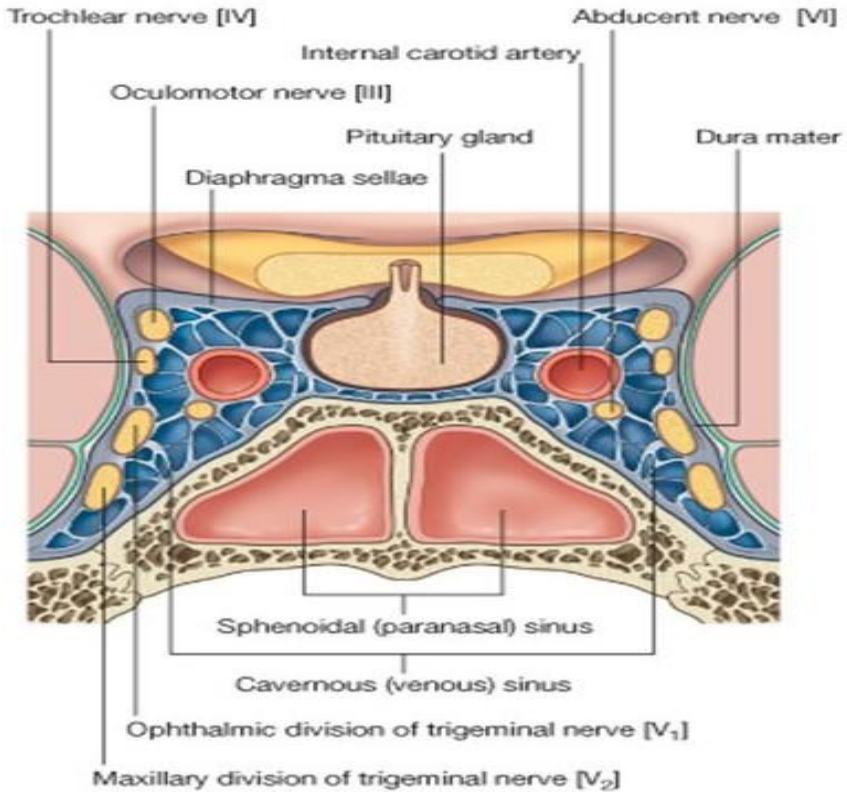
Sinus's anterior and posterior walls are called outer and inner frontal table, respectively. The inner table is a relative thin bony plate that separates frontal sinus from the anterior cranial fossa posteriorly. On the other hand, the outer table is a considerable thick bony wall. On the posterior wall (inner table) of the sinus, there are venous drainage channels called "foramina of Breschet." These foramina have clinical significance in their role in spreading the infection from the sinus toward intracranially. Also, these foramina act as sites of mucosal invagination within the bone, so failing to completely remove the mucosa in these sites during the sinus obliteration procedure may predispose to the development of mucocele. The floor of each frontal sinus forms the anterior roof of the orbit. The floor consisted of a thin bone which can be eroded by the mucocele. Frontal sinus ostium is located at the posteromedial part of sinus's floor. The frontal sinus drainage pathway has an hour-glass shape, with the narrowest point of this pathway corresponds to "the frontal beak" which represents the frontal sinus ostium.

Blood supply, innervation and lymphatic drainage

Frontal sinus receives blood supply from the supratrochlear and supraorbital arteries (branching from ophthalmic artery). Venous drainage is by the superior ophthalmic and diploic veins. Lymph drainage is across the face to the submandibular nodes. Frontal sinus receives innervation from the supratrochlear and supraorbital nerves.

Sphenoid sinus

Sphenoid sinuses occupy the body of sphenoid bone. Classically, there are two asymmetrical sinuses separated by off-midline intersphenoid bony septum.



Pic. 16. Sphenoidal sinus and cavernous sinus

The sphenoid sinus is bounded by the internal carotid artery, optic nerves, cavernous sinus, and sella turcica. Sphenoid sinus drains into the sphenoethmoidal recess through a single sphenoid ostium in the sinus's anterior wall, which opens medial to superior turbinate.

Blood supply, innervation, and lymphatic drainage

Arterial supply is from the posterior ethmoidal artery and posterior septal artery. Veins drain via the posterior ethmoidal vein to the superior ophthalmic vein. The sinus mucosa receives innervation from the posterior ethmoidal nerve and the orbital branch of pterygopalatine ganglion. The Lymph drains to the retropharyngeal nodes.

Rhinitis

Rhinitis describes inflammation of the nasal mucosa but is clinically defined by symptoms of nasal discharge, itching, sneezing and nasal blockage or congestion.

Chronic rhinitis can be classified into allergic, non-allergic and infective.

I Allergic rhinitis
A Intermittent
B Persistent
II Non-allergic rhinitis
A Vasomotor rhinitis
1 Irritant triggered
2 Cold air
3 Exercise (eg, running)
4 Undetermined or poorly defined triggers
B Gustatory rhinitis
C Infectious
1Acute
2 Chronic
III Occupational rhinitis
A Caused by protein and chemical allergens, IgE-mediated
B Caused by chemical respiratory sensitizers, immune mechanism uncertain
C Work-aggravated rhinitis
IV Other rhinitis syndromes
A Hormonally induced
1 Pregnancy rhinitis
2 Menstrual cycle related
B Drug-induced
1 Rhinitis medicamentosa
2 Oral contraceptives
3 Antihypertensives and cardiovascular agents

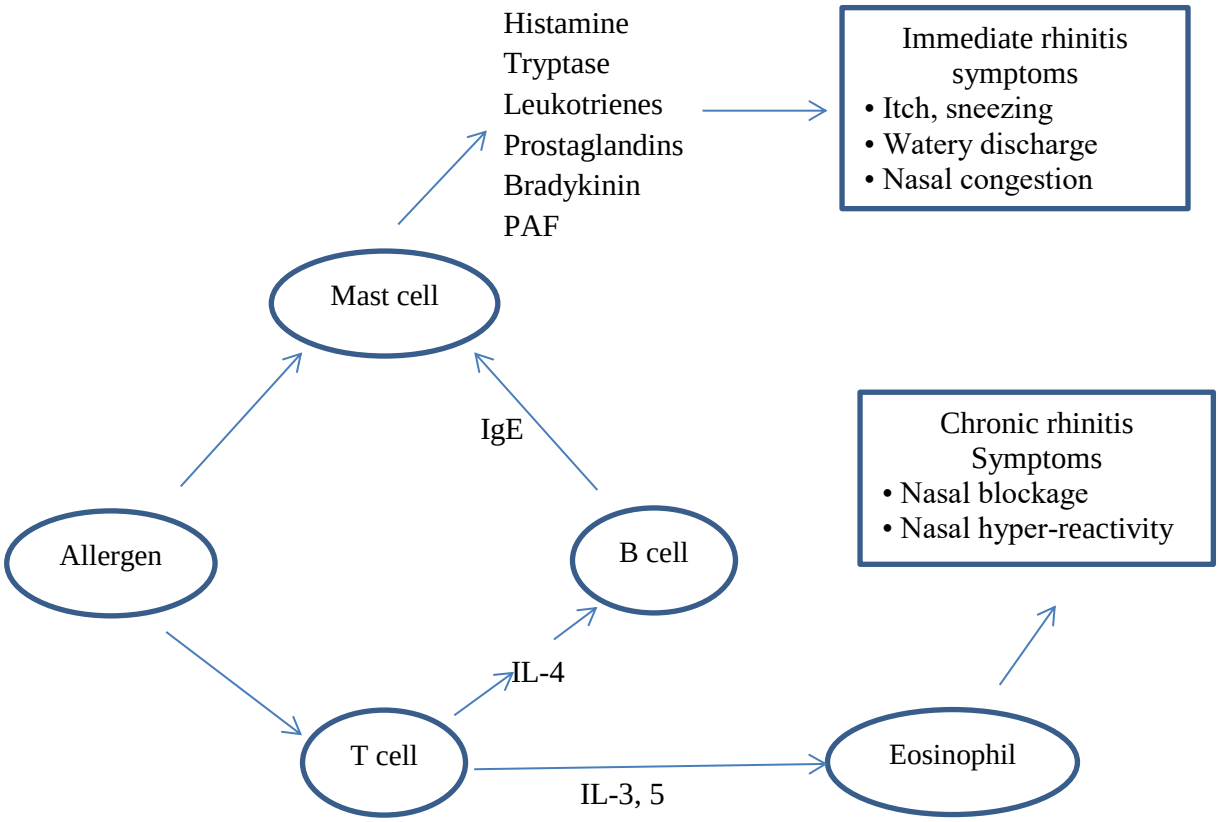
4 Aspirin/NSAIDs
5 Other drugs
C Atrophic rhinitis
D Rhinitis associated with inflammatory-immunologic disorders
1 Granulomatous infections
2 Wegener granulomatosis
3 Sarcoidosis
4 Midline granuloma
5 Churg-Strauss
6 Relapsing polychondritis
7 Amyloidosis
Conditions that may mimic symptoms of rhinitis
A Nasal polyps
B Structural/mechanical factors
1 Deviated septum/septal wall anomalies
2 Adenoidal hypertrophy
3 Trauma
4 Foreign bodies
5 Nasal tumors
6 Choanal atresia
7 Cleft palate
8 Pharyngonasal reflux
9 Acromegaly (excess growth hormone)
C Cerebrospinal fluid rhinorrhea
D Ciliary dyskinesia syndrome

Allergic rhinitis

Allergic rhinitis (AR) is a symptomatic disorder of the nose, induced after allergen exposure by an IgE-mediated inflammation of the nasal mucosa.

Pathophysiology – numerous inflammatory cells, including mast cells, CD4-positive T cells, B cells, macrophages and eosinophils infiltrate the nasal lining upon exposure to an allergen, and release cytokines (interleukin 3, 4, 5, 13) that promote IgE production by

plasma cells. IgE production, in turn, triggers the release of mediators, such as histamine and leukotrienes that are responsible for arteriolar dilation, increased vascular permeability, itching, rhinorrhea, mucous secretion and smooth muscle contraction. The mediators and cytokins released during the early phase of an immune response to an inciting allergen, trigger a futher cellular inflammatory response over the next 4 to 8 hours (late-phase inflammatory response) which results in recurrent symptoms.



Pic. 17. Mechanism of allergic rhinitis

Symptoms of allergic rhinitis include rhinorrhea, nasal obstruction, nasal itching and sneezing, which are reversible spontaneously or with treatment.

The new classification of AR is based on symptomps and quality-of-life parameters. Durations of symptoms is subdivided into "intermittent" or "persistent" disease, while severity is subdivided into "mild" or "moderate-severe", depending on symptoms and quality of life.

Intermittent	**Persistent**
≤ 4 days per week	**> 4 days per week**
or ≤ 4 weeks	**and > 4 weeks**
Mild	**Moderate-Severe**
• normal sleep	• abnormal sleep
• no impairment of daily activities, sport, leisure,	• impairment of daily activities, sport, leisure
• normal work and school	• abnormal work and school
• no troublesome symptoms	• troublesome symptoms

Treatment:

- avoidance measures
- oral/nasal antihistamines
- intranasal/systemic corticosteroids
- leukotriene receptor antagonists
- allergen immunotherapy

Oral H1-antihistamines

• first-generation antihistamines, e.g. chlorphenamine, diphenhydramine, cause sedation and reduce academic and/or work performance and should be avoided.

• second-generation antihistamines, e.g. acrivastine, cetirizine, desloratadine, fexofenadine, levocetirizine, loratadine and mizolastine are less sedating

• do not cause significant QT prolongation at normal therapeutic doses

H1-antihistamines – topical nasal

• Azelastine therapeutic effects superior to oral antihistamines for rhinitis symptoms.

• Do not improve symptoms due to histamine at other sites, such as the eye, pharynx, lower airways and skin.

• Fast onset of action within 15 min – useful as rescue therapy.

Topical intranasal corticosteroids

• Meta-analysis shows that intranasal corticosteroids are superior to antihistamines.
• Act by suppression of inflammation at multiple points in the inflammatory cascade.
• Reduce all symptoms of rhinitis
• Systemic absorption negligible with mometasone and fluticasone, modest for the remainder and high for betamethasone and dexamethasone – these should be used short term only.

Systemic glucocorticosteroids

• Rarely indicated in the management of rhinitis, except for: severe nasal obstruction short-term rescue medication for uncontrolled symptoms on conventional pharmacotherapy important social or work-related events, e.g. examinations, weddings
• Oral corticosteroids should be used briefly and always in combination with a topical nasal corticosteroid.

Anti-leukotrienes

• Anti-leukotrienes are of two kinds: (i) receptor antagonists (LTRAs, e.g. montelukast and zafirlukast) and (ii) synthesis inhibitors, e.g. zileuton .
• There is a spectrum of individual responsiveness to LTRAs that is currently not predictable.

Allergen immunotherapy

Allergen immunotherapy involves the repeated administration of an allergen extract in order to reduce symptoms and the need for rescue medication on subsequent exposure to that allergen. Immunotherapy can be highly effective and is the only treatment that is able to modify the natural history of AR and offer the potential for long-term disease remission.

Vasomotor rhinitis

Vasomotor rhinitis, a type of non-allergic rhinitis, may be episodic or perennial. The exact pathophysiology of vasomotor rhinitis has never been established, and for this reason, it is often classified as ‘‘idiopathic’’ rhinitis. When rhinorrhea is the predominant symptom, there appears to be enhanced cholinergic

glandular secretory activity based on the fact that atropine like agents effectively reduce secretions.

Gustatory rhinitis

Gustatory rhinitis (rhinitis symptoms associated with eating) is a form of non-allergic rhinitis felt to be vagally mediated that may respond to intranasal anticholinergic agents. Patients with predominant nasal congestion may have nociceptive neurons that have heightened sensitivity to stimuli such as temperature change, airborne irritants, foods (especially hot and spicy foods), alcoholic beverages, cold dry air, and exercise.

Occupational rhinitis

Occupational rhinitis may be triggered by allergic factors, such as laboratory animal antigen or irritant factors, such as chemicals, grain dust, and ozone. Irritant exposures elicit neutrophilic inflammation in the nasal mucosa, whereas allergic exposures are associated with eosinophils, basophils, eosinophilic cationic protein, and tryptase in the nasal lavage.

Pregnancy and menstrual cycle rhinitis

Symptoms of rhinitis during pregnancy and at the time of patients' menstrual cycles have long been considered to be hormonally induced. The most common causes of nasal symptoms during pregnancy are allergic rhinitis, sinusitis, rhinitis medicamentosa, and vasomotor rhinitis. A type of rhinitis unique to the pregnant patient is ''vasomotor rhinitis of pregnancy'' or ''pregnancy rhinitis.'' Pregnancy rhinitis had been defined as rhinitis without an infectious, allergic, or medication-related cause that starts before the last 6 weeks of pregnancy (corresponding to 34 weeks gestation), persists until delivery, and resolves completely within 2 weeks after delivery.

Drug-induced rhinitis

Drug-induced rhinitis may be caused by ACE inhibitors, a-receptor antagonists used in the treatment of benign prostatic hypertrophy, and phosphodiesterase-5 selective inhibitors used for treatment of erectile dysfunction. Aspirin and other NSAIDs may produce rhinorrhea as an isolated symptom or as part of aspirin-exacerbated respiratory disease.

Rhinitis medicamentosa

Rhinitis medicamentosa may develop after the repetitive and prolonged use of topical a-adrenergic nasal decongestant sprays such as oxymetazoline and phenylephrine. Patients may develop rebound congestion, tachyphylaxis, reduced mucociliary clearance because of loss of ciliated epithelial cells, and on rare occasions, nasal septal perforation. The pathophysiology of this condition is not understood.

Treatment of rhinitis medicamentosa consists of suspending the use of topical decongestants and administering intranasal corticosteroids to control symptoms while allowing the rebound effects of the nasal decongestant spray to resolve.

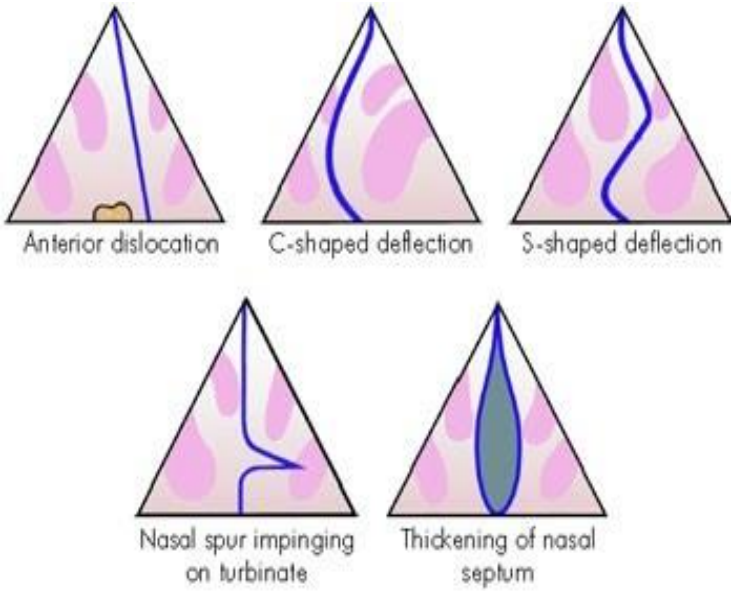
Atrophic rhinitis

Primary (idiopathic) atrophic rhinitis is a chronic condition characterized by progressive atrophy of the nasal mucosa, nasal crusting, nasal dryness (caused by atrophy of glandular cells), and fetor. The nasal cavities appear abnormally wide on examination, and there is absence of identifiable turbinates on sinus CT, referred to as the ‘‘empty nose syndrome.’’ Secondary atrophic rhinitis is most commonly a result of chronic sinusitis or excessive surgery to the nasal turbinates. Although saline irrigation is the mainstay of treatment, topical or systemic antibiotics are indicated with the appearance of purulent nasal secretions.

Deviated Nasal Septum

Introduction The nasal septum comprising of bony and cartilaginous parts separates the nasal cavity into right and left sides both anatomically and physiologically. It is an accepted fact that some amount of deviation of nasal septum is common and having a perfectly straight septum is a rarity. Nasal septum deviation has no relation to age, sex or ethnicity.

Etiology: Developmental, traumatic, compensatory septal deviation may occur.



Pic. 18. Deviated nasal septums

Classification of deviated nasal septum:
1 C shaped
2 S shaped
3 Caudal dislocation
4 Spur
5 Thick septum (reduplication)

Cotte's classification:

1 Simple - mild deflection that does not obstruct.

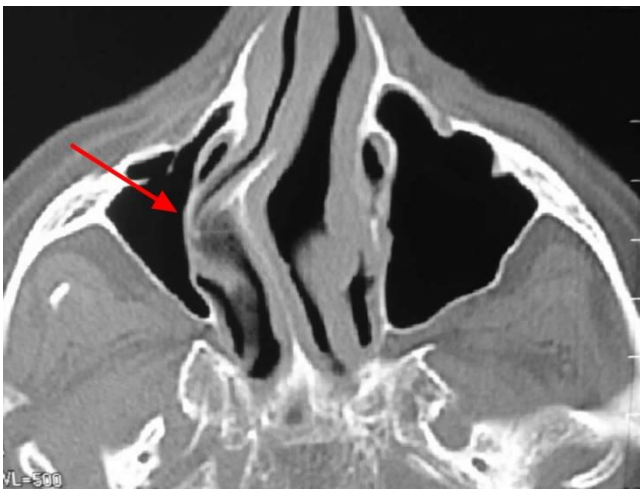
2 Obstructed - severe deflection touching lateral wall but shrinking on vasoconstriction.

3 Impacted - marked angulations of the septum with a spur in contact with the lateral wall even application of a vasoconstrictor.

Deviated nasal septum can be asymptomatic in an individual or may cause nasal obstruction and symptoms of rhinosinusitis like nasal discharge, facial pain, epistaxis, disturbance of smell. The pathology in sinonasal cavity can also affect the functioning of throat and ear.

In the case of nasal obstruction, nose breathing can change to mouth breathing, resulting in facial changes with an increase in lower facial height and transverse maxillary deficiency. In addition, in patients with septal deviation a compensatory hypertrophy of the inferior nasal concha bone can be found in the right and left sides.

Changes in the nasal area and air way can lead to significant changes in craniofacial growth.



Pic. 19. Deviated nasal septum - CT scan image

Treatment options Simple deviations with mild deflection of the septum which does not cause obstruction do not require any treatment. More severe deviations with or without impaction on the lateral wall of the nose is managed by surgical treatment. The essential indication of septal surgery being skeletal septal obstruction (bony and cartaginous).

Septal surgeries for correction of deviated nasal septum:

1 Sub mucous resection

2 Septoplasty

- Conventional
- Endoscopic
- Laser assisted
- Radio frequency assisted
- Extra-corporeal
- Powered endoscopic nasal septal surgery

Epistaxis

Epistaxis is a very common presenting complaint and the most common emergency for the Otolaryngologist Head and Neck Surgeon. All causes of nasal bleeding can be divided into two groups: local and systemic factors.

Local factors:

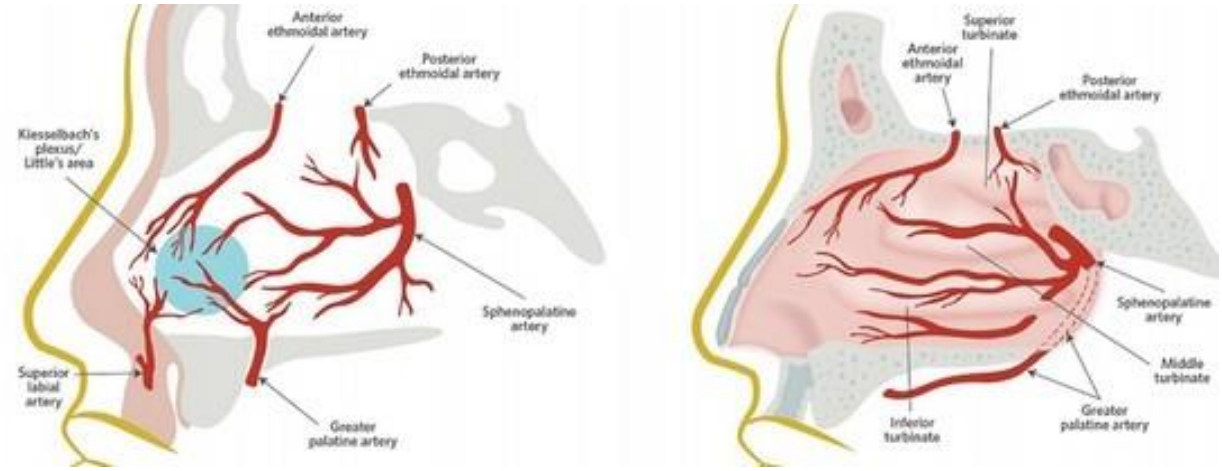
1. injuries of the nose and foreign bodies in the nasal cavity

2. Inflammation
3. low relative humidity
4. anatomical deformity and tumors of the nasal cavity, barotrauma, use of nasal spray (especially steroid)

Systemic factors:

- colds,
- allergies
- arterial hypertension (persistent increase in blood pressure)
- blood diseases (leukemia, hemophilia),
- vascular diseases (purpura),
- systemic connective tissue disease,
- heart failure,
- deficiency of vitamin K or C.
- side effect of some medications (most often a non-steroidal anti-inflammatory drugs type of aspirin, ibuprofen and anti-coagulants)
- use of alcohol.
- increased pressure in superior vena cava) due to mitral stenosis or superior mediastinal tumors.

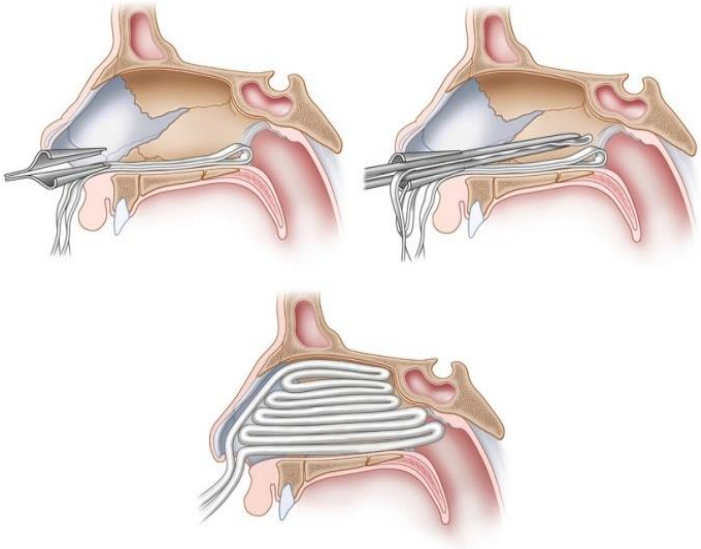
Epistaxis can be divided into anterior and posterior based upon the arterial supply and location of the offending vessel. Most cases of epistaxis are anteriorly located (90-95%), and are usually treated effectively after visual localization with local chemical or electrical cauterization via anterior rhinoscopy.



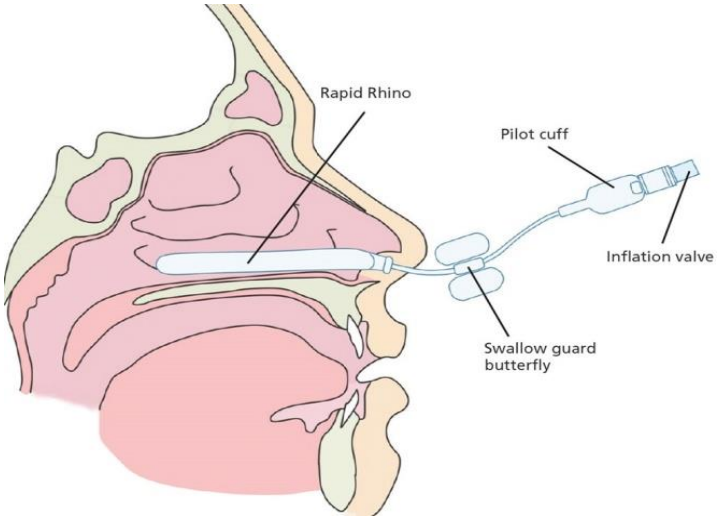
Pic. 20 Epistaxis illustration: vascular supply of the nasal septum and lateral nasal wall

Initial (‘‘first-line’’) treatment can include combinations of direct nasal compression, application of topical agents including vasoconstrictors, cautery of the bleeding site with chemicals or electrocautery, or packing with a variety of resorbable and nonresorbable materials.

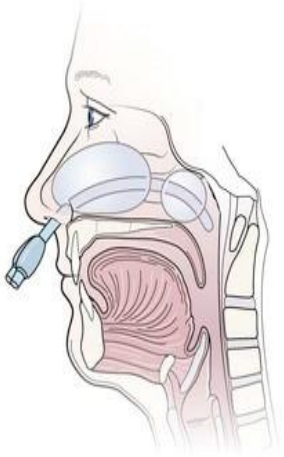
Anterior nasal packing is recommended in case if the exact origin of bleeding cannot be identified on nasal endoscopy. Can be removed with 24 hour and can be kept upto 2-3 days. Multiple devices and materials are available.



Pic. 21. Anterior nasal packing with horizontal layers (nasal speculum, forceps, gauze)

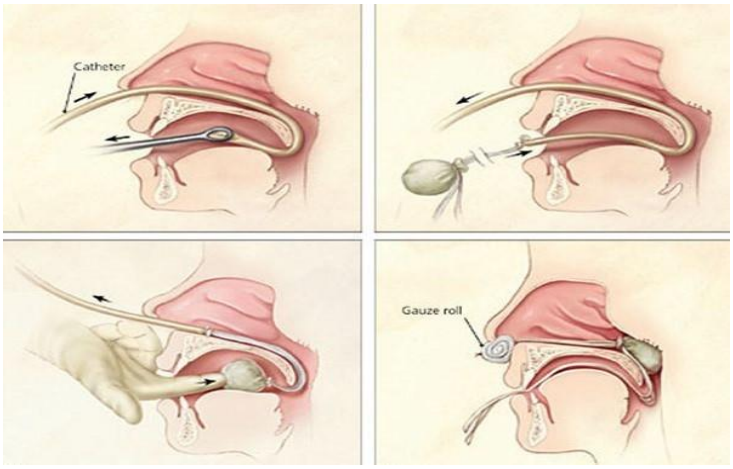


Pic. 22. Anterior nasal packing with a Rapid Rhino



Pic. 23. Anterior and posterior nasal packing with epistaxis balloon

Posterior nasal packing - packing of the nose and nasopharynx usually for cases of posterior epistaxis, most often involves nonresorbable packing materials.



Pic. 24. Posterior nasal packing with posterior pack

Cautery may be performed with topical administration of chemically active agents, such as silver nitrate (25%-75%), chromic acid, or trichloroacetic acid, or through the application of heat or electrical energy, typically electrocautery or‘‘hot wire’’ thermal cautery. Sites for application of cautery can range from the small anterior septal vessels in Kiesselbach’s plexus to named larger arteries, such as the sphenopalatine artery and its branches located posterior in the nose. Cautery should be performed with direct view of the target bleeding site to prevent excessive tissue injury and increase chances of success. Complications from cautery include infection, tissue injury, and possibly septal necrosis and resultant perforation.

Surgical Arterial Ligation. Transnasal sphenopalatine artery ligation and transnasal endoscopic sphenopalatine artery ligation (TESPAL). TESPAL is now the most commonly employed surgical arterial ligation technique, with a reported success rate of up to 98%. Traditionally, ligation of the anterior and posterior ethmoid arteries required an open approach and dissection along the medial orbit. Transnasal endoscopic anterior ethmoid artery ligation requires additional steps, such as preoperative computed tomography imaging to confirm anterior ethmoid arterial anatomy and endoscopic accessibility, and consideration of additional unique complications, such as cerebrospinal fluid leak and orbital injury. Endovascular embolization of the anterior and/or posterior ethmoid arteries is contraindicated, as they arise from the ophthalmic artery with inherent risks of blindness with such a procedure.

Endovascular embolization of epistaxis was first described by Sokoloff et al in 1974 with use of small gelfoam particles. Since that time, embolization has been refined with advancement of microcatheters and development of embolic materials, such as polyvinyl alcohol particles and calibrated embolic particles. Endovascular embolization is best suited for posterior nosebleeds, and current practice involves embolization of the bilateral spheno-palatine/distal internal maxillary arteries. Despite use of meticulous techniques and knowledge of external carotid-internal carotid anastomoses, blindness and stroke are the most feared complications of embolization.

Nasal bone fractures

Introduction: Nasal bone fractures are common because:

1. Nose happens to be the most prominent portion of the face
2. Increasing number of road traffic accidents
3. Increasing incidence of domestic violence
4. Increase in the number of individuals taking part in contact sports

Fractures involving nasal bones if not properly and promptly treated leads to:

1. Nasal deformities

2. Intranasal dysfunction like nasal block

Fracture nasal bone is known to cause higher incidence of morbidity and complications when compared that of fractures involving other facial bones.

In order to treat this condition properly it is necessary to accurately diagnose this condition by:

1. Looking for crepitus and tenderness over the nasal bone area
2. Radiographic evaluation of nasal bones.

ENT doctors are more interested in knowing:

1. Location of fracture site (like sidewall, dorsum, or the entire nasal bone)
2. To know whether the fracture involves the right nasal bone / left nasal bone or both sides
3. Whether there is any displacement of the fractured fragments (medial/lateral)
4. To identify the presence of concurrent fractures to other facial bones/nasal septum. When there is the presence of fractures involving other facial bones/ severe fractures of nasal septum it is prudent to perform open reduction.

Pathophysiology

1. Nasal bones and underlying cartilage are susceptible for fracture because of their more prominent and central position in the face.
2. These structures are also pretty brittle and poorly withstands force of impact.
3. The ease with which the nose is broken may help protect the integrity of the neck, eyes, and brain. Thus it acts as a protective mechanism.
4. Nasal fractures occur in one of two main patterns - from a lateral impact or from a head-on impact. In lateral trauma, the nose is displaced away from the midline on the side of the injury, in head-on trauma, the nasal bones are pushed up and splayed so that the upper nose (bridge) appears broad, but the height of the nose is collapsed (saddle-nose deformity). In both cases, the septum is often fractured and displaced.
5. The nasal bone is composed of two parts: a thick superior portion and a thin inferior portion. The intercanthal line demarcates these two portions. Fractures commonly occur below this line.

6. Nasal bones undergo fracture in its lower portion and seldom the upper portion is involved in the fracture line. This is because the upper portions of the nasal bone is supported by its articulation with the frontal bone and frontal process of maxilla.

7. Because of the close association between nasal bone and the cartilaginous portions of the nose, and the nasal septum it is quite unusual for pure nasal bone fractures to occur without affecting these structures. If closed reduction alone is performed to reduce nasal bone fractures without correction of nasal septal fractures, this could cause progressive nasal obstruction due to uncorrected deviation of nasal septum. This is because of the tendency of the nasal septum to heal by fibrosis which causes bizarre deviations like "C" "S" etc.

Stranc-Robertson classification:

Type I injury: Fractures due to this type of injury does not extend behind the imaginary line drawn from the lower end of nasal bone to the anterior nasal spine. In this type of injury the brunt of the attack is borne by lower cartilaginous portion of the nasal cavity and the tip of the nasal bones. This type of injury may cause avulsion of upper lateral cartilages, and occasionally posterior dislocation of septal and alar cartilages.

Type II injury: This type of injury involves the external nose, nasal septum and anterior nasal spine. Patients with this type of injury manifest with gross deviations involving the dorsum of the nose including splaying of nasal bones, flattening of dorsum of nose and loss of central support of the nose.

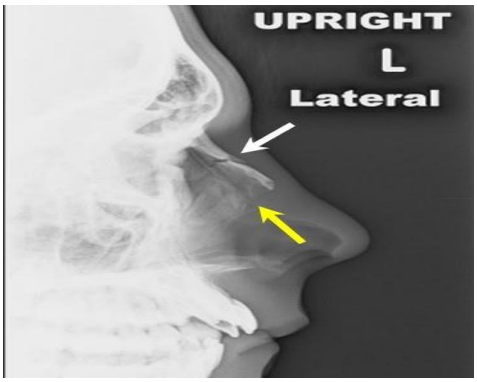
Type III injury: This injury involves orbit and intracranial structures.

Clinical examination: depression or displacement of nasal bones, edema of nose, epistaxis, fractures of septal cartilage with displacement or mobility, crepitus on palpation.

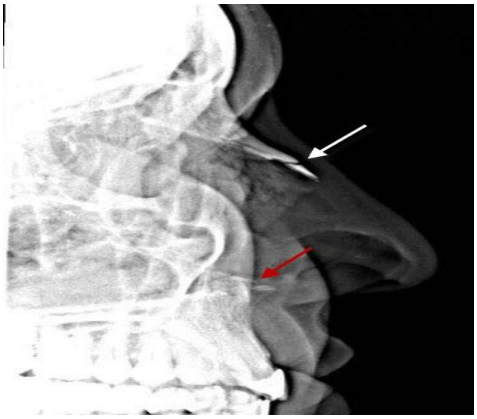
Radiology:

X-ray of nasal bone has very minimal role in the diagnosis of fractures involving the nasal bones. CT scan of nose and sinuses helps in identifying fractures involving other facial bones and in Lefort II and Lefort III fractures. Ultrasound using 10 MHz probe gives a clear view of the nasal bone area thereby facilitating easy identification of fractures. It also has the advantage of nil radiation hazard to the patient.

Radiology: X-ray of nasal bone, CT scan of nose and sinuses helps in identifying fractures.



Pic. 25. There is a transverse fracture of the nasal bone (white arrow). The longitudinal line (yellow arrow) does not represent a fracture



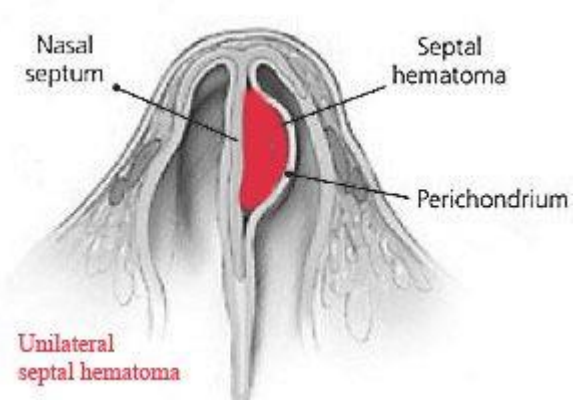
Pic. 26. Enhanced lateral radiograph shows a transverse fracture of the nasal bone (white arrow). There is also an associated fracture of the anterior nasal spine (red arrow)

Management: If fractures of nasal bones are left uncorrected it could lead to loss of structural integrity and the soft tissue changes that follow may lead to both unfavourable appearance and function. Reduction is ideally performed immediately after injury before oedema sets in. If oedema has already set in it is prudent to wait for it to subside because it is difficult to ascertain adequacy of reduction in the presence of oedema.

1. Closed reduction
2. Open reduction
3. Conservative management

Nasal septal hematoma

Introduction Nose is the most commonly injured facial structure. Most nasal injuries do not require immediate intervention, but trauma is resulting in septal hematoma is an exception. A nasal septal hematomais a rare but serious complication of nasal or facial trauma.



Pic. 27. Septal hematoma

It refers to the collection of blood under the mucoperichondrium or mucoperiosteum of nasal septal cartilage or bone.

Etiology A septal hematoma usually occurs secondary to nasal trauma. The latter can be in the form of sports injuries, road-side accidents, falls, assault or occupational injuries. Iatrogenic septal hematoma may arise as a complication of nasal surgeries like septal correction, endoscopic sinus surgery or turbinate surgery. Atraumatic septal hematoma is rarely seen in patients with bleeding diathesis or as an adverse effect of antiplatelet/anticoagulant drugs.

Pathophysiology The exact mechanism underlying the formation of nasal septal hematoma remains controversial. Septal cartilage is an avascular structure, 2 mm to 4 mm thick, which receives its nutrients supply from the overlying perichondrium. Physicians hypothesize that trauma results in sharp buckling forces that pull the closely adherent mucoperichondrium from the underlying cartilage. This causes the rupture of submucosal vessels which ultimately causes collection of blood between the cartilage and the perichondrium. Hematoma thus formed, results in pressure related ischaemic changes and the subsequent necrosis of the septal cartilage. If the trauma is severe enough, the septal cartilage gets fractured, and

blood sweeps to the opposite side resulting in a bilateral septal hematoma. This situation is more hazardous as it doubles the compromise on the nutrient supply of septal cartilage and hastens the process of cartilage necrosis. Hematoma acts as an ideal medium for bacterial proliferation and colonization. If left untreated, it gets infected within 72 hours leading to the formation of a septal abscess.

History and Physical A nasal hematoma usually presents within the first 24 to 72 hours after trauma. The most common symptom is the nasal obstruction which can be either unilateral or bilateral depending on the type of hematoma. Other symptoms include pain, rhinorrhea, and fever. In many cases, symptoms are non-specific. Therefore, a high index of suspicion should be kept especially if a patient presents with nasal deformity and/or nasal pain following trauma. Also if a post-traumatic nasal obstruction does not resolve with a local vasoconstrictive agent or blood clot removal, the possibility of nasal hematoma should be strongly considered.

Clinical examination: asymmetry of the septum with bluish or reddish mucosal swelling suggests a hematoma. A hematoma feels soft and fluctuant in contrast to deviated nasal septum which will be firm and concave on the opposite side. Another important feature of septal hematoma is the lack of reduction in size on the application of decongestant sprays like oxymetazoline 0.05%.

Evaluation Usually clinical diagnosis. Rarely CT or MRI might be considered if the diagnosis is equivocal on physical exam.

Treatment / Management A nasal septal hematoma should be drained urgently to avoid undue complications. The procedure is done under local anesthesia. Larger hematomas are drained by incising the mucosa over the most fluctuant area. The incision is given in the anteroposterior direction parallel to the nasal floor. In case of bilateral hematoma, a staggered incision is made to avoid through and through the septal perforation. Systemic antibiotics are prescribed to prevent serious, infective complications.

Complications are bound to occur in untreated or improperly treated hematomas. A septal abscess is the most common acute complication of septal hematoma. If left untreated, the infection can spread to the nearby anatomical structures like paranasal sinuses, orbit or intracranial structures, through the venous draining the mid-face. Avascular necrosis and secondary infection can lead to the collapse of

septal cartilage causing various types of nasal deformities. In children, destruction can cause an altered growth of mid-face and permanent facial deformity.

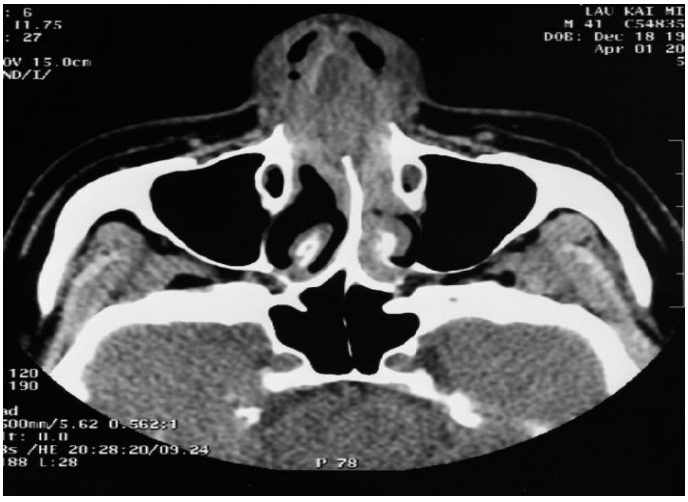
Nasal septal abscess

Introduction Nasal septal abscess (NSA) is defined as a collection of pus between the cartilaginous or bony nasal septum and the mucoperichondrium or mucoperiosteum.

Etiology It's an uncommon condition that most commonly develops in a pre-existing septal hematoma with usually a history of nasal trauma. NSA is also less frequently associated with nasal furunculosis, sinusitis, influenza, dental infection, and after nasal septal surgery. The most common etiological agent is Staphylococcus aureus, others less frequently include Streptococcus pneumoniae, group A beta hemolytic Streptococcus, anaerobes, Hemophilus influenzae.

History and Physical The most common presentation of the NSA is nasal obstruction and pain. In contrast, septal hematoma usually presents as painless nasal obstruction after injury. Other signs and symptoms include headache, fever, saddle nose, and swelling of the nasal septum.

Evaluation Usually clinical diagnosis. Rarely CT or MRI might be considered if the diagnosis is equivocal on physical exam. CT scan with contrast enhancement is very helpful for the diagnosis, the involved area and cartilage status.



Pic. 28. Computer tomographic scan: Axial computed tomography image showing swelling of the nasal septum with hypodense fluid collection

Treatment / Management Emergency surgical drainage of nasal septal abscess and intravenous antibiotic administration is required. The abscess can be drained by a horizontal incision from behind forwards across the swollen area, as low as possible in the septum. Bilateral drainage is only necessary if the abscess is bilateral with an intact septal cartilage, so that cartilage still exists between the collections of purulent material on both sides. It is sometimes advantageous to remove a piece of mucoperichondrium to facilitate drainage.

Complications The accumulation of pus between the cartilage and perichondrium will lead to ischemia and necrosis of the cartilage; this may result in septal cartilage destruction, saddle nose deformity, functional dysfunction and cosmetic problems. In children in particular, these consequences may be affect the normal development of the nose and maxilla. Life-threatening intracranial complications, such as brain abscess, meningitis and cavernous sinus thrombosis; especially in the immunocompromised patients may progress rapidly if the NSA remains untreated.

Paranasal sinus fractures

Anatomy and pathology Traumatic forces directed at the facial skeleton is transmitted and absorbed by a framework of facial bones that surround paranasal sinuses. Disruption to the bony framework results in paranasal sinus fractures. The Le Fort classification system categorizes common types of bony framework disruption along "lines of weakness" and can be associated with certain types of paranasal sinus fractures. In addition to Le Fort classification system, other classifications have been devised for facial fractures that are too complex for the Le Fort fracture lines which focus on anatomical subunits of the mid-face including zygomaticomaxillary complex (ZMC), the naso-orbitoethmoidal complex (NOE), and the dentoalveolar complex (DAC).

Le Fort I and II fractures, ZMC fractures: associated with maxillary sinus fracture (most common paranasal sinus fracture).

Le Fort II and III fractures: associated with ethmoid sinus fracture.

NOE fractures: associated with frontal/ethmoid sinus fracture.

The commonly used classification is as follows:

Le Fort type I:

• horizontal maxillary fracture, separating the teeth from the upper face

• fracture line passes through the alveolar ridge, lateral nose and inferior wall of the maxillary sinus

• also known as a Guerin fracture.

Le Fort type II:

• pyramidal fracture, with the teeth at the pyramid base, and nasofrontal suture at its apex

• fracture arch passes through the posterior alveolar ridge, lateral walls of maxillary sinuses, inferior orbital rim and nasal bones

• uppermost fracture line can pass through the nasofrontal junction or the frontal process of the maxilla

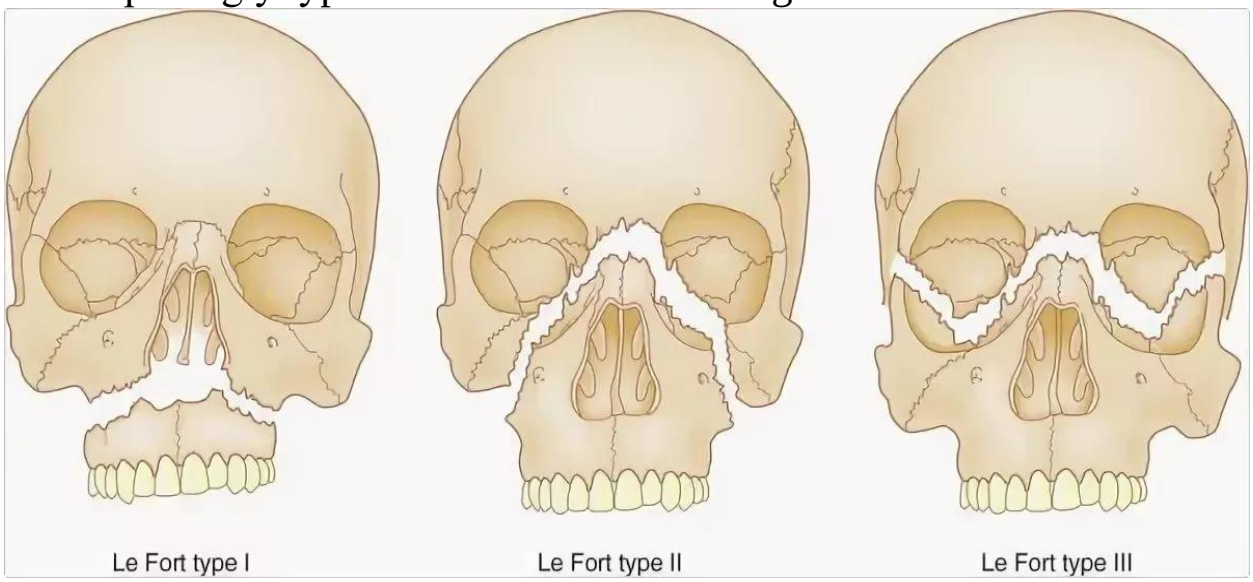
Le Fort type III

• craniofacial disjunction

• transverse fracture line passes through nasofrontal suture, maxillo-frontal suture, orbital wall, and zygomatic arch/zygomaticofrontal suture

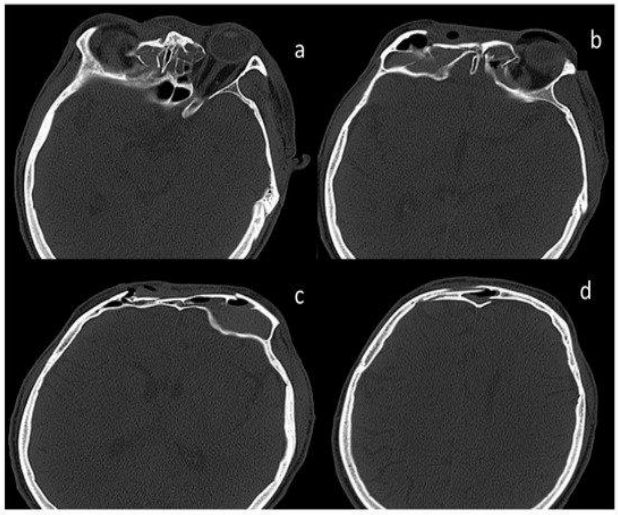
• because of the involvement of the zygomatic arch, there is a risk of the temporalis muscle impingement

• unsurprisingly type III fractures have the highest rate of CSF leak



Pic. 29. Le Fort fractures

Naso-orbito-ethmoid (NOE) fractures associated with anterior and posterior frontal sinus wall fractures are among the most challenging cranio-maxillofacial injuries. These represent a major emergency, having a potentially severe clinical picture, with intracranial hemorrhage, cerebrospinal fluid (CSF) leak, meningeal lesions, pneumocephalus, contusion or laceration of the brain matter, coma, and in some cases death.

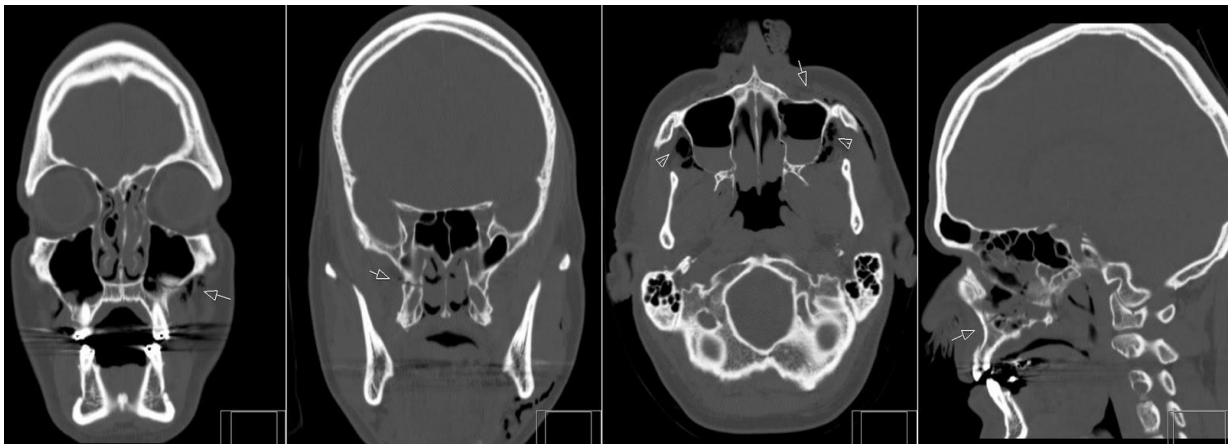


Pic. 30. CT images, bone window, axial sections. (a) Naso-orbito-ethmoid (NOE) fracture with major depression and ethmoid hemosinus, (b,c) comminuted fracture of the anterior frontal sinus walls with frontal hemosinus, subcutaneous emphysema, and left pneumo-orbitis, (d) displaced posterior frontal sinus wall fracture

Maxillary sinus fractures are most commonly caused by blunt force trauma to the face. The mechanism of injury varies based on the age of the patient, the external force vector, and anatomic location. Trauma can come from motor vehicle accidents (MVA's), domestic disputes, falls, industrial accidents, or assaults with or without a weapon. Injuries to the lateral midface are more common than to the anterior midface.

Evaluation Though physical examination may be enough to diagnose obvious MSFs, many times, concurrent imaging will be needed to exclude other injuries and assist with presurgical planning. Computed tomography (CT) of the maxillofacial bones without contrast is the gold standard imaging study needed in facial trauma patients. Computed tomography will reveal cortical fractures and show the amount of bony displacement, which will help to dictate

further management. This can also help identify other local associated fractures, which will help dictate further consults, treatment plans, and prognosis. CT scans are also excellent for identifying foreign bodies, local hemorrhage, and some soft tissue injuries. Less commonly used imaging modalities, including magnetic resonance imaging (MRI), plain film x-ray, and ultrasound, which have limited utility compared to non-contrast CT. With that in mind, MRIs tend to be indicated as a compliment to initial CT scans, specifically with suspected CSF leaks and orbital soft tissue injuries. MRIs are also better able to detect and differentiate a herniation of orbital fat versus entrapment of orbital muscle. Ultrasound imaging has been used with some success for superficial fracture diagnosis in the orbit, specifically with the zygomatic arch and nasal bones.



Pic. 31. Radiology managememt

Surgical indications for MSFs are poor facial aesthetics, restoration of midfacial height, correction of malocclusion, and repair of midface projection. If surgical indications are met, basic treatment of the anterior sinus wall should be based around repositioning of the fracture segments with fixation using plates, screws, mesh grafts, or resorbable foils. Most surgeons will plan for open reduction and internal fixation (ORIF) via a Caldwell-Luc (gingival buccal) incision or an endoscopic nasal approach.

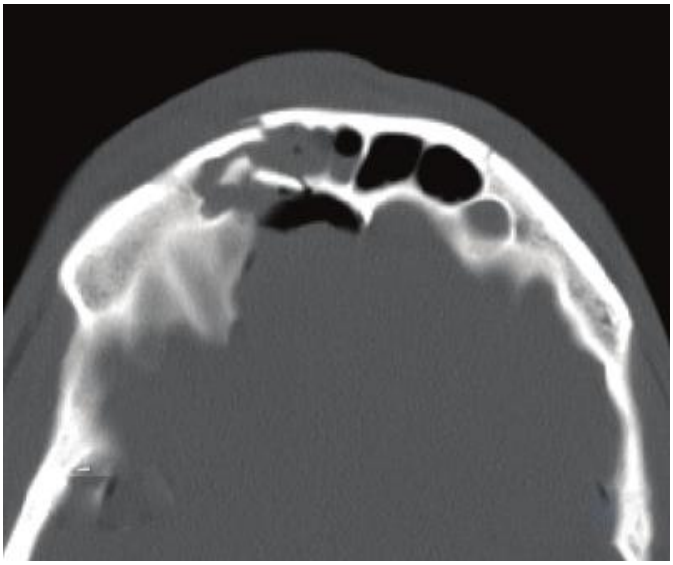
Differential diagnosis: zygomatic arch fracture, mandible fracture, inferior orbital rim fracture, orbital floor fracture, nasal bone fracture, nasoorbitoethmoid fracture, acute, chronic, or recurrent maxillary rhinosinusitis, facial hematoma or contusion.

Complications: extraocular eye muscle entrapment (inferior rectus and inferior oblique muscles), orbital cellulitis, orbital abscess, orbital emphysema enophthalmos, hypophthalmos, maxillary sinusitis, maxillary sinus mucocele, epistaxis, CSF rhinorrhea, oromaxillary fistula, dental malocclusion, facial asymmetry, aesthetic concerns, pressure sensitivity, chronic facial pain, infraorbital nerve, paresthesia.

Frontal sinus fracture is a common phenomenon due to road traffic accident or direct head trauma. There are a myriad of short- and long-term potential complications associated with this type of injury that may involve not only the frontal sinuses, but more importantly the brain. The anterior wall of the frontal sinus is thick and resistant to injury. It requires greater force to fracture than any other facial bone. The most common frontal sinus fractures involve a combination of the anterior and posterior tables with or without frontal recess involvement.

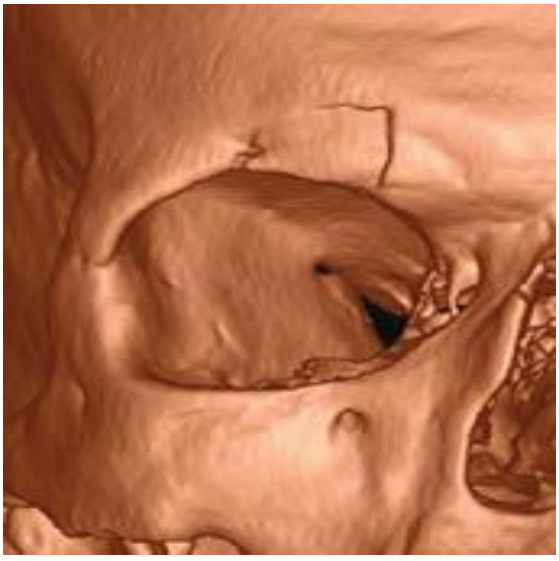
Clinical evaluation: periorbital ecchymosis and pain in the forehead region. Blunt or penetrating trauma to this region can also produce altered sensation of the forehead and scalp resulting from injury to the supraorbital and supratrochlear neurovascular bundles. If the patient is seen shortly after the injury, one might also appreciate a depression in the bony forehead. This finding is seldom seen because of the soft tissue swelling. Crepitation might be palpable in evaluating such bony injuries. The examiner should always be cognizant of other concomitant injuries such as intracranial bleeding and cervical injuries. A focused exam of the frontal sinus should include evaluation for any contour deformity and/or frontal lacerations and neurosensory deficits. Evaluation of the nose for possible cerebrospinal fluid (CSF) is imperative in patients with frontal sinus fractures. If the posterior table of the sinus or the base of the skull in the region of the cribriform plate is fractured, the dura might be lacerated, causing leakage of CSF into the sinus and then into the middle meatus. Confirmation of the CSF from the nose can be done at bedside by performing the double halo test or ring test. A small sample of the nasal drainage is placed on a cotton sheet; if a red central ring (blood) surrounded by a clear halo (CSF) is observed, the practitioner should suspect leakage of CSF. Other confirmatory tests include sending samples of the fluid to the laboratory for evaluation of chloride and glucose and the β2-transferrin test. CSF will usually have high

chloride and low glucose concentration compared with serum. Presence of β2-transferrin, a protein marker found exclusively in CSF. Examination of deep wounds should be performed under sterile technique, as these can be through and through injuries.



Pic. 32. Axial CT images can be used to document anterior and posterior table injuries

Radiographic features, CT: if a fracture is visualized care should be taken to assess whether the anterior table (between sinus and scalp), posterior table (between sinus and dura) or both are involved, and to note the degree of displacement. Presence of pneumocephalus, particularly if intradural (subarachnoid) is important as it increases the risks of subsequent CSF leak, meningitis and mucocoeles due to trapped mucosal elements.



Pic. 33. 3-D reconstruction can be used to document the spatial orientation of the displaced bone fragments

Classification:

1. Frontal recess involvement

A frontal recess injury involves the floor of the frontal sinus and the outflow tract. It may also involve the anterior skull base.

2. Anterior table fractures

Fractures of the anterior table of the frontal sinus vary from minimally displaced to severely displaced/comminuted depending upon the severity of the trauma and size of the sinus.

Minimal displacement

Less severe trauma may result in fractures of the anterior table which are minimally or nondisplaced.

Moderate displacement

As the traumatic force increases, fracture segments are displaced more significantly.

Severe comminution

The most severe anterior table injuries result in comminution of the anterior table bone.

3. Posterior table fracture

Posterior table fractures carry a higher risk of intracranial injury because they create a communication with the intracranial space.

Small displacement

Less severe posterior table injuries may result in minimal or no displacement.

Large displacement

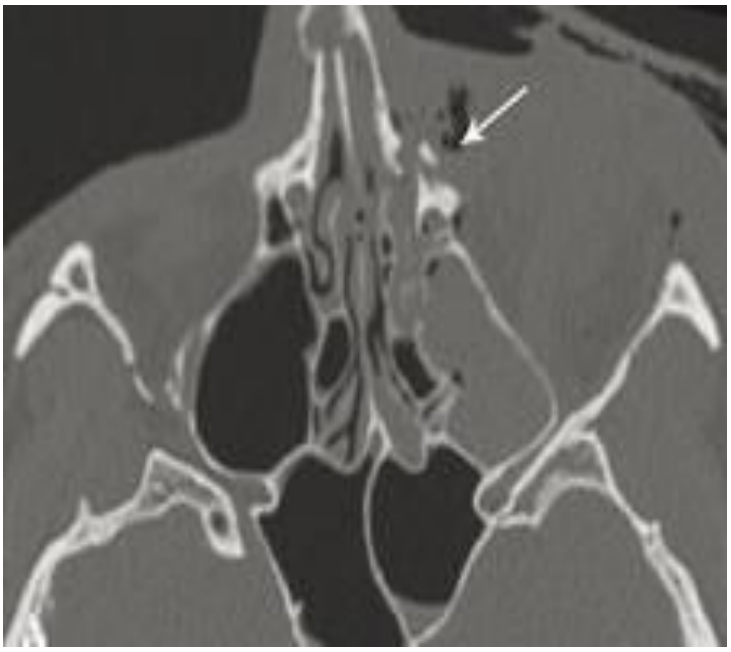
More severe injuries often result in displacement and/or comminution of the posterior table.

Moderate to severe comminution

The most severe injuries result in severe comminution of both the anterior and posterior tables, with an increased risk of intracranial injury.

Treatment Indications for treatment include cosmetic deformity (a depressed outer table fracture can be seen as an indentation of the forehead), meningitis or sinus dysfunction (e.g. development of a mucocoele). The treatment goals: (a) repair of the defect and elimination of the conduit from the intracranial space to the outside and (b) elimination of any CSF pressure gradient that may develop across the surgical repair.

Ethmoidal sinus fracture



Ethmoidal sinus fractures are usually seen in the setting of nasal fractures (naso-orbitoethmoid complex fractures). Commonest mechanism of injury - motor vehicle accident. Common fracture site is superior sinus wall (cribriform plate), isolated injury rare, usually associated with injury to NOE complex and frontal sinus.

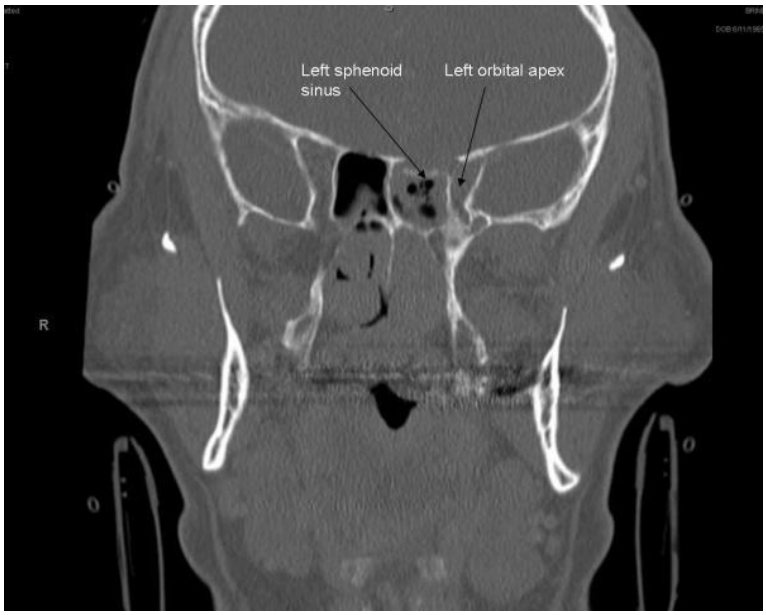
Pic. 34. Ethmoidal sinus fracture

Specific symptoms: hyposmia, anosmia

Complications: olfa ctory nerve damage, dacryocystitis, CSF rhinorrhea, and intracranial infection.

Sphenoidal sinus fracture

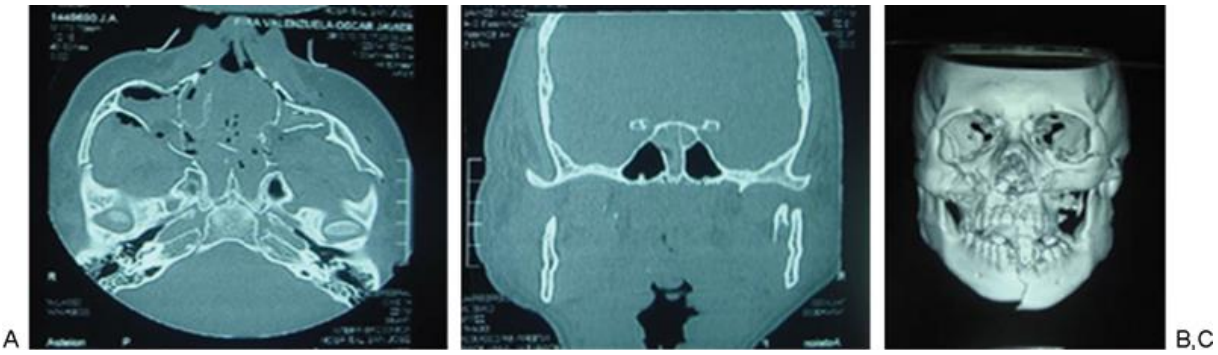
Transsphenoidal fractures are usually seen in the setting of more extensive base of skull fractures, often extending to involve anterior cranial fossa or petrous temporal bones. Mechanism of injury various,



isolated injury rare, usually associated with complex craniofacial injuries. Specific symptoms: clear nasal discharge, visual disturbance.

Radiologic diagnosis of skull base fractures is usually obtained by a high-resolution CT scan.

Pic. 35. Sphenoid wall fractures



Pic. 36. High-energy trauma and panfacial fractures. (A) Zygomatic arch, malar, and upper jaw fractures. (B) Sphenoid sinus fracture. (C) Computed tomography three-dimensional reconstruction showing the fractures at the middle third and jaw

Complications: optic/oculomotor nerve damage, internal carotid artery damage, CSF rhinorrhea.

Sphenoid bone and sphenoid sinus fractures have a high morbidity due to the complexity of trauma and soft tissue involved at the level of the brain. Skull base fractures are of high importance in neurotrauma.

Sinusitis

Sinusitis, defined as inflammation of one or more of the paranasal sinuses, is characterized as acute when lasting less than 4 weeks (ARS), subacute when lasting 4 to 8 weeks, and chronic when lasting longer than 12 weeks. Recurrent sinusitis consists of 4 or more episodes of acute sinusitis per year.

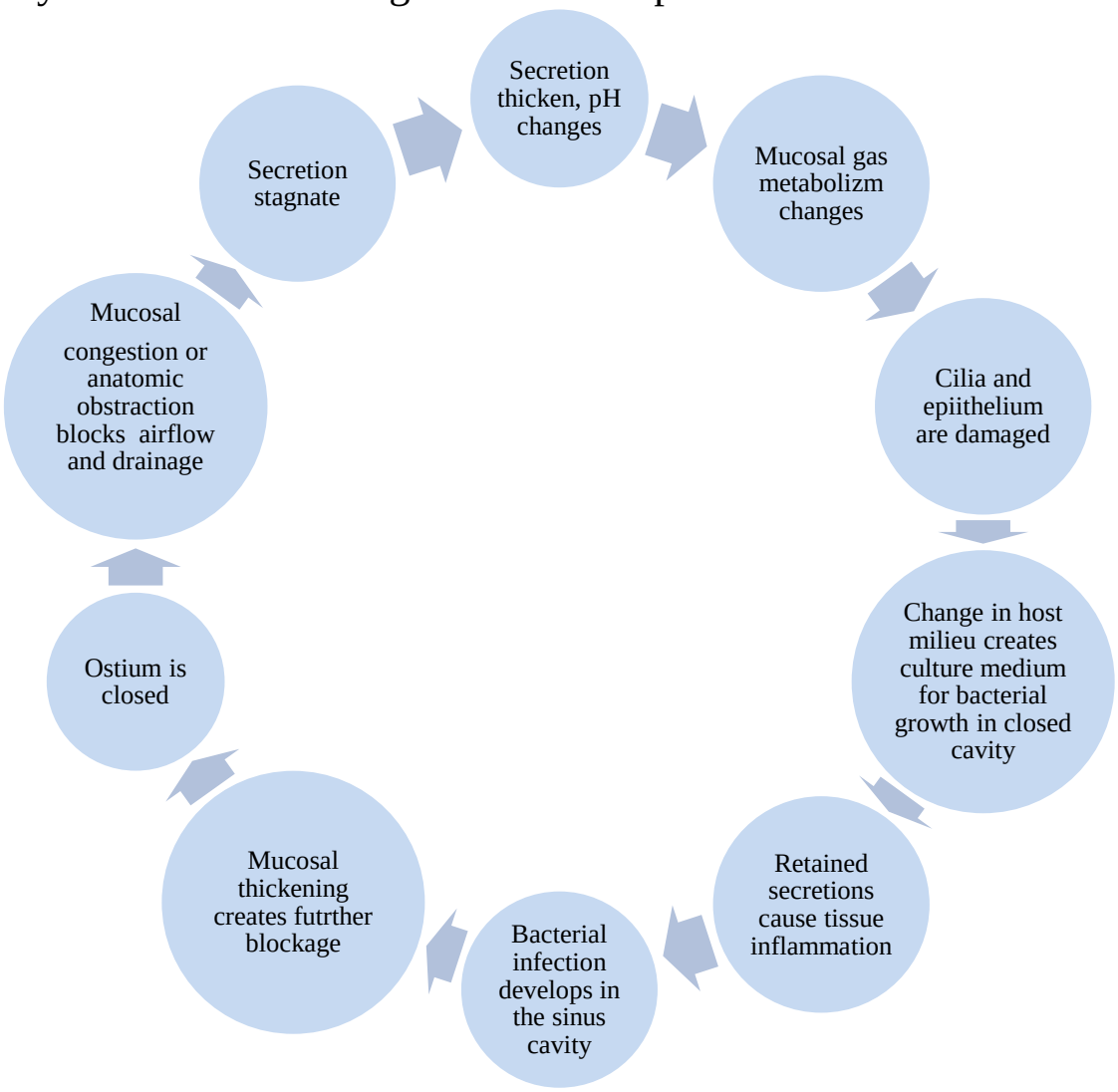
ARS may be classified further by presumed etiology, based on symptoms and time course, into acute bacterial rhinosinusitis (ABRS) or viral rhinosinusitis (VRS).

Acute sinusitis may originate from or be perpetuated by local or systemic factors predisposing to sinus ostial obstruction and infection. These factors include anatomic or inflammatory factors leading to sinus ostial narrowing, disturbances in mucociliary transport, and immune deficiency. Sinus ostial narrowing may be caused by acute viral upper respiratory infection or chronic allergic inflammation.

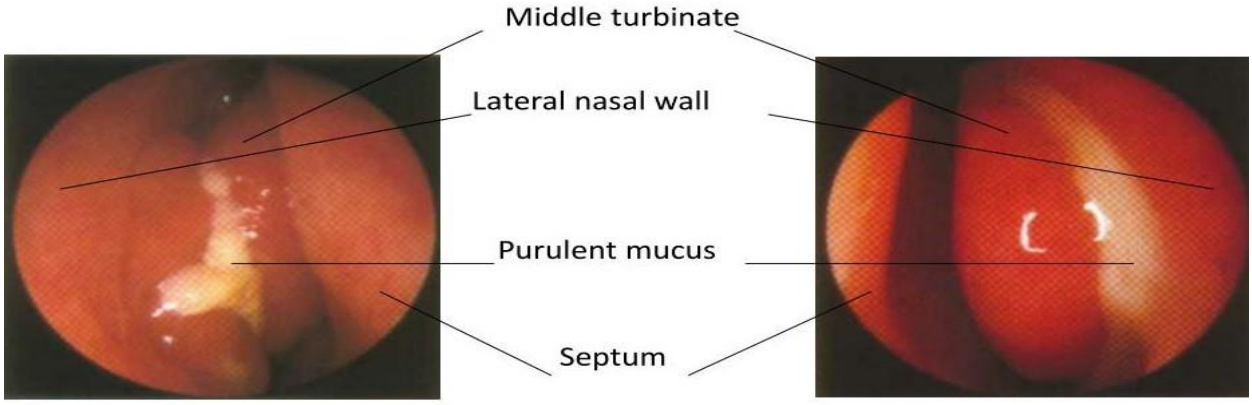
Several anatomic variants that may predispose to ostiomeatal narrowing, including Haller’s cells (infraorbital ethmoid cells), agger

nasi cells (the most anterior superior insertion of the middle turbinate), paradoxical curvature of the middle turbinate, bulla ethmoidalis with apparent medial contact, deformities of the uncinate process, and concha bullosa deformity (pneumatization of the middle turbinate).

In acute sinusitis, the predominant organisms are Streptococcus pneumoniae, Hemophilus influenzae, and (in children) Moraxella catarrhalis. Acute rhinosinusitis is diagnosed when a patient presents with up to 4 weeks of purulent (not clear) nasal drainage accompanied by nasal obstruction, facial pain-pressure-fullness, or both. Additional signs and symptoms of ABRS include fever, cough, fatigue (malaise), reduced sense of smell (hyposomia), lack of the sense of smell (anosmia), maxillary dental pain, and ear fullness or pressure, intranasal examination with anterior rhinoscopy or nasal endoscopy may demonstrate findings of unilateral purulent rhinorrhea or edema.



Pic. 37. Sinus cycle leading to rhinosinusitis



Pic. 38. Endoscopic examination of the left/right nasal cavity and left/right middle turbinate demonstrating purulent fluid in the middle meatus

Management of VRS is primarily directed toward relief of symptoms. Antibiotics are not recommended for treating VRS since antibiotics are ineffective for viral illness and do not provide direct symptom relief. Therefore, palliative medications such as analgesics, anti-inflammatory agents, nasal saline, decongestants, antihistamines, mucolytics, cough suppressants, and topical or oral corticosteroids may be used alone or in varying combinations for symptom relief.

Symptomatic relief of ABRS: analgesics, saline irrigation, topical nasal steroids, antibiotics.

Odontogenic sinusitis

Etiology Odontogenic sinusitis is most commonly the result of iatrogenic injury of the mucoperiosteum, or Schneiderian membrane, of the maxillary sinus. Dental procedures such as dental extractions, maxillary dental implant placement, sinus augmentation grafts (“sinus lift”), misplaced foreign bodies as well as orthognathic and cleft surgery procedures have all been associated with odontogenic sinusitis. Other potential etiologies include periodontal and periapical disease. Endodontic infections are typically the result of extension of dental caries into the dental pulp resulting in pulpitis and apical infection. Alternatively, chronic periodontitis may occur in the setting of chronic infection of a tooth socket. The resultant inflammation and/or disruption of the Schneiderian membrane leads to mucosal inflammation and altered mucociliary function within the maxillary sinus. Impaired mucociliary function results in altered mucus transport, impaired mucosal defenses, blockage of sinus ostia and

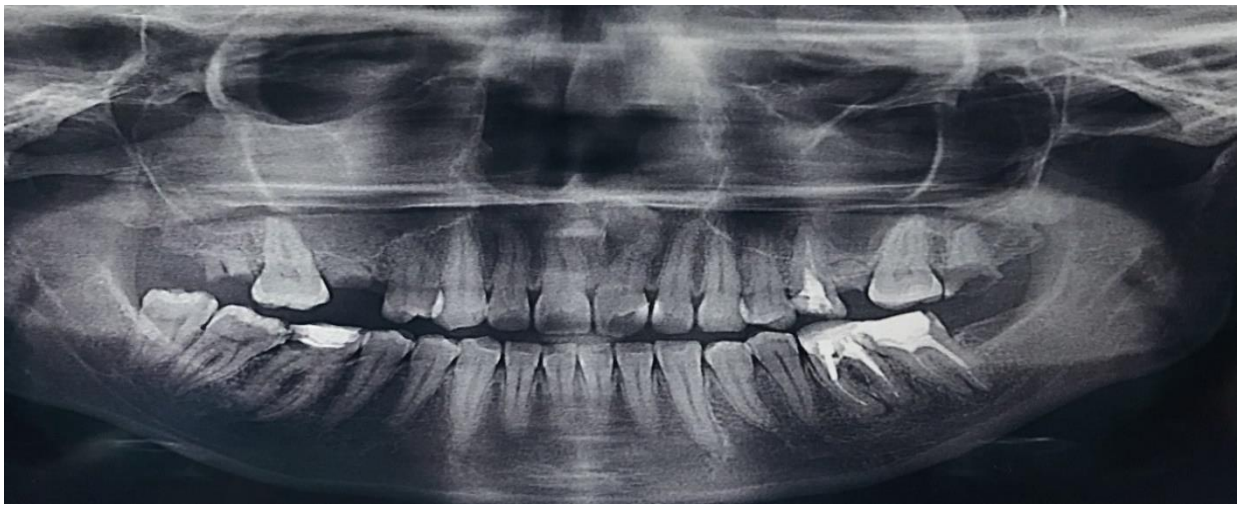
resultant bacterial infection and inflammation. Other less common etiologies of odontogenic sinusitis include maxillary bone trauma, odontogenic cysts, neoplasms or other inflammatory processes.

Microbiology Odontogenic sinus infections are generally polymicrobial with predominantly anaerobic organisms present in cultures, commonly including Peptostreptococcus, Prevotella, and Fusobacterium.

The clinical presentation of odontogenic sinusitis varies, but most commonly includes symptoms of facial pain or pressure, postnasal drip, nasal congestion, purulent anterior rhinorrhea that may be unilateral, foul smell or taste.

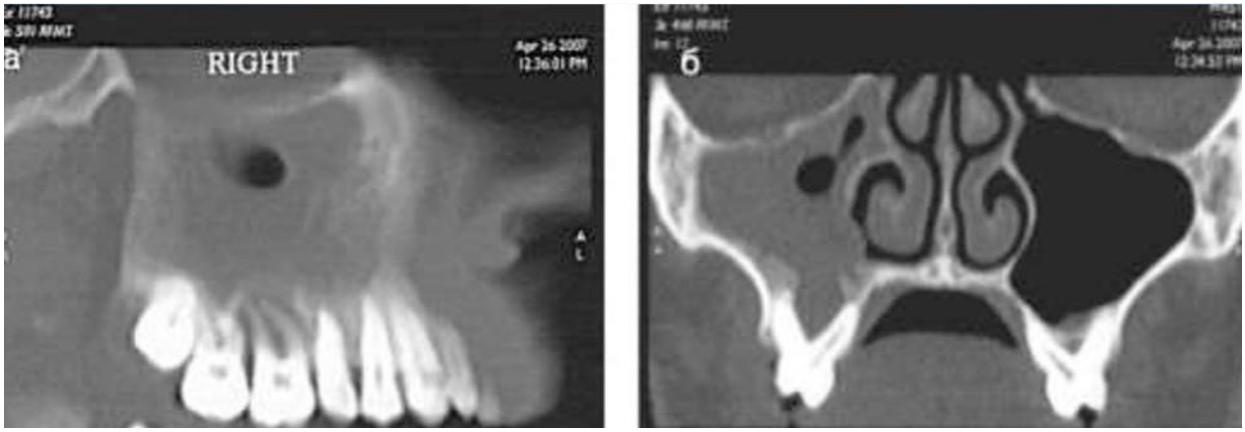
Intranasal examination with anterior rhinoscopy or nasal endoscopy may demonstrate findings of unilateral purulent rhinorrhea or edema.

Radiographic imaging is an essential diagnostic tool in the diagnosis and management of odontogenic sinusitis. Standard dental radiographs include periapical radiography and panoramic radiography.



Pic. 39. Panoramic radiography

Maxillofacial CT scans allow for a detailed examination of the patient’s paranasal sinus anatomy and detection of sinonasal inflammation.



Pic. 40. Computerized tomography (CT) of a patient with odontogenic sinusitis demonstrating complete opacification of the right maxillary with associated involvement of the ostiomeatal unit

Treatment Successful management of odontogenic sinusitis involves a combination of medical treatment, dental surgery and/or endoscopic sinus surgery.

Chronic Rhinosinusitis (CRS)

Introduction The nasal disorder of CRS is a chronic condition of the upper airway characterized histologically by the infiltration of inflammatory cells like eosinophils or neutrophils of the paranasal sinuses and nasal cavity. Clinically, nasal polyposis have nasal obstruction, hyposmia, rhinorrhea and reduced quality of life. Nasal polyps (NP) derived from the middle meatus are inflammatory outgrowths of paranasal sinus mucosa, most often benign, frequently bilateral, and typically develop in adulthood, and are characterized by inflammation. They present an abnormal remodeling response and a lack of immunoregulation, creating an imbalance and, consequently, favoring inflammation.

CRS is defined as inflammation of the nose and paranasal sinuses characterized by the presence of two or more of the following symptoms for greater than 12 weeks duration: nasal blockage/obstruction/congestion; nasal discharge; facial pain/pressure; reduction or loss of smell. Confirmation of the diagnosis of CRS with nasal polyps (CRSwNP) or CRS without nasal polyps (CRSsNP) is made by nasal endoscopy or sinus CT scan. The endotype complement of a CRS patient is defined by a set of interleukins (ILs), cytokines, growth-factors, and immunological inducers. Clusters are

identified based on analysis of IL-5, IFN-γ, IL-17A, TNF-α, IL-22, IL-1β, IL-6, IL-8, cationic eosinophilic protein, myeloperoxidase, TGF-β1, IgE, staphylococcus aureus-specific IgE for enterotoxin, and albumin in CRS in the nose itself and are highly modified with the disease. CRS can be classified into primary or secondary disease based on the extent of anatomical involvement and endotype dominance.

Physical examination: anterior rhinoscopy, oropharyngoscopy, nasal endoscopy, CT, MRI, bacterioscopy, nasal cytology, biopsy, mucociliary function, allergological assessment, olfaction assessment, laboratory tests.

Treatment: successful management of chronic rhinosinusitis involves a combination of medical treatment and endoscopic surgery.

Mucoceles of paranasal sinuses

A mucocele is defined as mucous filled epithelium lined sac. Mucoceles commonly involve ethmoidal and frontal sinuses. Mucoceles are commonly caused due to obstruction to drainage channel of paranasal sinuses. These expansile cystic masses are sometimes filled with mucopurulent secretions. Sometimes associated bone destruction is also evident.

Mucoceles are rather common in frontal sinuses. Next comes the ethmoidal sinuses. Isolated mucoceles involving ethmoidal sinuses are rather rare. They always occur in combination with frontal / sphenoid mucoceles.

Etiopathogenesis: mucoceles have been postulated to form due to obstruction of sinus ostia following chronic infections / allergic reactions involving paranasal sinuses. Previous trauma / surgery can also cause obstruction to sinus outflow channels causing formation of mucoceles. Ethmoiodal mucoceles if present in isolation could be caused by endoscopic ethmoidectomy. Paranasal sinuses continues to expand slowly owing to pent up mucous secretions. These mucoceles are lined by dilated ciliated columnar epithelium which secrete mucous causing expansion of the cyst. Continuing expansion of this cyst puts pressure on the bony walls of paranasal sinuses, causing

bony erosion and remodeling. Unchecked extension of sinus cavity can cause extension of mucocele into orbit, nasopharynx and cranial cavity. In addition to pressure changes inflammatory mediators like prostaglandins, interleukins and tumor necrosis factor present within mucoceles also contribute to their expansion capability. Three main theories of pathogenesis of mucocele formation has been postulated:

1. Pressure erosion
2. Cystic degeneration of glandular tissue
3. Active bone resorption and regeneration

Frontal mucoceles have been classified into 5 types depending on its extent.

Type I: In this type the mucocele is limited to the frontal sinus only with or without orbital extension.

Type II: Here the mucocele is found involving the frontal and ethmoidal sinuses with or without orbital extension.

Type IIIa: In this type the mucocele erodes the posterior wall of the frontal sinus with minimal or no intracranial involvement.

Type IIIb: In this type the mucocele erodes the posterior wall with major intra cranial extension.

Type IV: In this type the mucocele erodes the anterior wall of the frontal sinus.

Type Va: In this type there is erosion of both anterior and posterior walls of frontal sinus without or minimal intracranial extension.

Clinical features: usually patients with mucoceles involving paranasal sinuses don't have nasal / sinus symptoms. Only symptoms they present with are ophthalmological in nature.

1. Pain: This is commonly periorbital in nature. This is caused by inflammation and stretching of nasal and sinus mucosa, rarely from dura. Pain is usually transmitted by trigeminal nerve.
2. Progressive proptosis
3. Visual disturbances (diplopia) / blurring of vision: This is caused by erosion of the bony casing around optic nerve. Any further expansion of mucocele will cause compression of optic nerve compromising its blood supply. Infections from mucocele can reach the optic nerve when the bony casing around the optic nerve is breached by the enlarging mucocele.
4. Epiphora

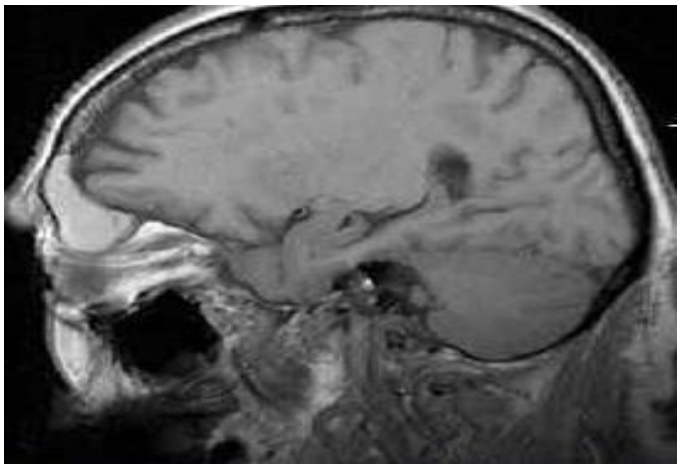
5. Impaired ocular mobility

Role of radiology in diagnosis of mucoceles:

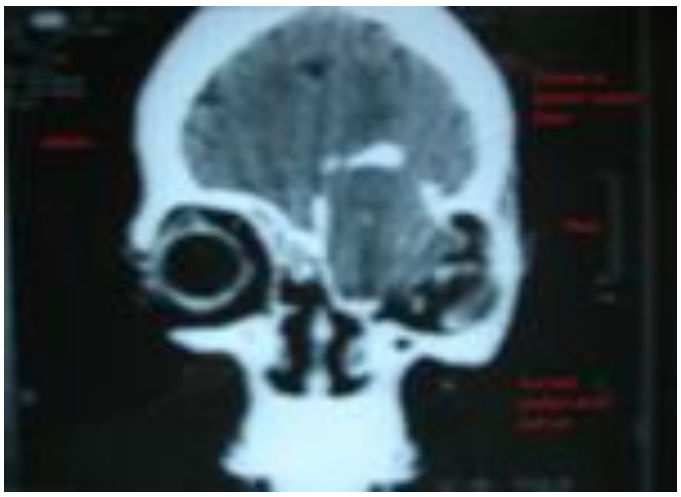
Radiological images of sinuses demonstrate thinning and expansion of affected paranasal sinus walls. Sinuses affected by mucoceles usually appears homogenous and airless. Plain x-ray of paranasal sinus mucoceles show the following features:

1. Soft tissue density mass seen obliterating sinuses
2. Expansion of paranasal sinus
3. Evidence of bone thinning and erosion

CT scan shows lesions with greater clarity. Precise extension of the lesion can be assessed by studying CT scan images. Scans reveal well defined expansile lesion with obliteration of paranasal sinus air cell cavities.



Pic. 41. Paranasal sinus mucocele



Pic. 42. Image showing coronal CT scan showing expansile lesion involving left frontal sinus with proptosis of left eye

Mucoceles are ideally managed surgically. Before the advent of endoscopic procedures, external frontoethmoidectomy was considered to be the ideal management modality.

Classification of surgical approaches:

I. Transnasal approach:

Endoscopic sinus surgery
Microscopic sinus surgery
Trans sphenoidal approach

II. External approaches:

Caldwel Luc approach
Osteoplastic frontal sinus surgery
External Ethmoidectomy
Lateral rhinotomy
Craniofacial resection

Endoscopic procedures are currently the commonly used surgical approach in managing mucoceles.

Complications of sinusitis

Introduction Complications of sinusitis can be classified as local or systemic. Local complications are mostly due to the anatomical proximity of the sinuses to the surrounding structures. The orbit and the skull base are the most closely related structures to the paranasal sinuses as they share same bony margins. Complications generally occur when the infection spread to these areas due to anatomic proximity.

Local complications of rhinosinusitis include mucocele, pre-septal cellulitis, orbital cellulitis, subperiosteal abscess, orbital abscess, osteomyelitis, meningitis, brain abscess, subdural empyema, and thrombosis of venous sinuses.

Complications of rhinosinusitis can be classified as orbital, intracranial or osseous.

Routes of spread

Sinus infections can spread through the lamina papyracea. Ethmoidal sinuses are separated from the orbit by a thin bone layer called the lamina papyracea. Besides being thin, this lamina has congenital dehiscences and perforating vessels and nerves. Infections in ethmoidal sinuses can spread through the natural dehiscence of this layer into the orbit especially in children.

The close neighborhood of the sinuses with the orbit is another way. The base of the frontal sinus forms the roof of the orbit. The frontal sinus may have bony dehiscences that form a route for the spread of the infections. Especially in adults, infections of frontal sinuses can lead to orbital complications.

Another route of spread is anatomic dehiscences and weaknesses of surrounding bone structures. Bacterial infections can spread through these bony barriers because of sclerosis in chronic sinusitis and osteolysis in acute sinusitis.

Roots of teeth form a transition route for infections. The maxillary sinus is closely related to the roots of the first upper molar and second premolar teeth. Infections of these roots can cause isolated maxillary sinusitis.

Venous connections and diploic veins are another major contagion mechanism. Diploic veins (veins of Breschet) are located in frontal sinuses and infections can spread by thrombophlebitis of these structures. Also, the veins between orbit and the sinuses have no valves, so the infections of sinuses are easily spread to the structures of the orbit.

The infraorbital canal is located at the base of the orbit and causes the spread of maxillary sinus infections to the orbit.

The infection can spread to the endocranium by contiguity from a frontal, ethmoidal or sphenoidal osteitis or osteomyelitis. It can also spread through retrograde septic thrombophlebitis in the diploic veins, favored by the absence of valves. Another manner for dissemination is the septic emboli which migrate by hematologic way.

Predisposing factors Immunodeficiency (e.g., HIV, malignancies), diabetes mellitus, and incomplete antibiotic treatments of sinusitis are the most frequent predisposing factors for complications.

Orbital Complications

The most common structure affected by sinus infections is orbit. Orbital complications arise most commonly from ethmoid sinusitis and rarely from sphenoid sinusitis. Routes of spread that are responsible for orbital complications are dehiscent lamina papyracea, open suture lines or venous connections. Symptoms related to orbital involvement are swelling, impairment in extraocular muscle movements, and exophthalmos. A direct or vascular spread of the infection may lead to periorbital or orbital cellulitis. Edema and

erythema in the eyelid are the first symptoms of periorbital cellulitis. Maxillary sinusitis causes swelling in the lower eyelid and frontal sinusitis in the upper eyelid. Since lamina papyracea is a thin bone layer separating the orbit from ethmoid sinuses and natural dehiscences are common, orbital complications mostly arise from ethmoid sinusitis. In younger individuals and children, ethmoidal sinusitis is the most common cause of orbital complications, whereas in adults, orbital complications mostly arise from infections in frontal sinuses. Also in adults, infections in sphenoid sinuses can spread to orbital nerve and may lead to blindness.

Pathogenesis

The roof of the orbit is surrounded by frontal sinus and the floor by maxillary sinus. The orbit is also bordered by ethmoid sinuses medially. Sometimes, orbital complications may be the first symptom of sinusitis.

Routes of spread to the orbit are bony dehiscence/defects (congenital/acquired), neurovascular foramens, and venous channels.

Lamina papyracea is a thin bone layer separating the ethmoid and maxillary sinuses from the orbit and infections can spread to the orbit by the natural dehiscences or by penetrating this bone layer.

The venous system in the orbit has no valves so the infection in the sinuses can also easily extend to orbit by the thrombophlebitis of these valveless veins. Infections in the sinuses can also spread directly to the orbit by neurovascular foramens (anterior and posterior ethmoid foramina).

Orbital complications are more common in children than adults because bony septa between sinuses and orbit are thinner; also, bones have open suture lines and large vascular foramens.

Classification of Orbital Complications

Classification of the orbital complications helps us to decide if medical treatment is enough or surgical intervention is needed.

Orbital complications have been categorized by Chandler et al. according to severity into five stages:

Stage I: Inflammatory edema and pre-septal cellulitis

Stage II: Orbital cellulitis

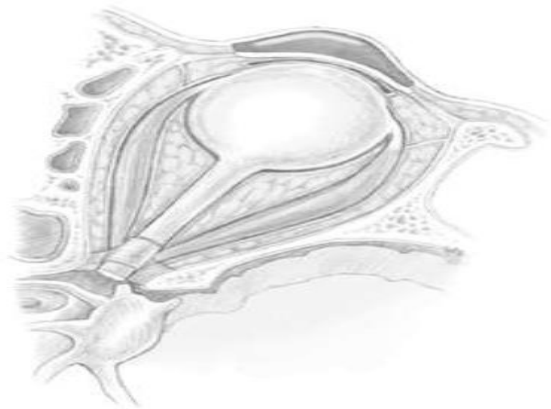
Stage III: Subperiosteal abscess

Stage IV: Orbital abscess

Stage V: Cavernous sinus thrombosis

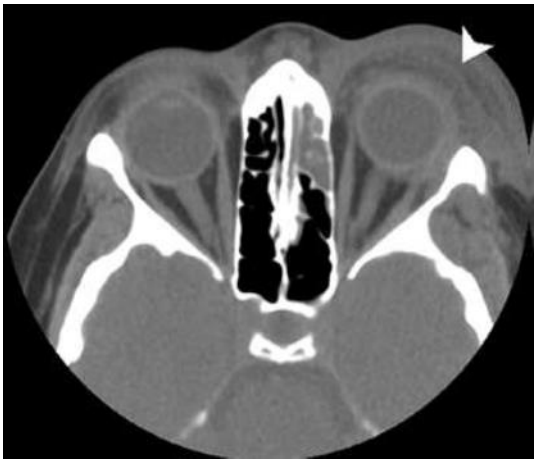
Stage I, Periorbital Cellulitis/Pre-septal Cellulitis

Orbital septum and tarsal plate play an important role in limiting the progression of the infection into the orbit. Pre-septal cellulitis is the inflammation or the infection of the skin around the eye and the eyelid, located in front of the orbital septum. This complication occurs when the ethmoid vessels around the skin were obstructed by inflammation due to the sinusitis. Patients with pre-septal cellulitis have edema and erythema around the eye and eyelids but no tenderness and no proptosis/chemosis. The movement of the eyeball and vision are normal. Also, these findings are used in the differential diagnosis of pre-septal and orbital cellulitis.



Pic. 43. Periorbital Cellulitis

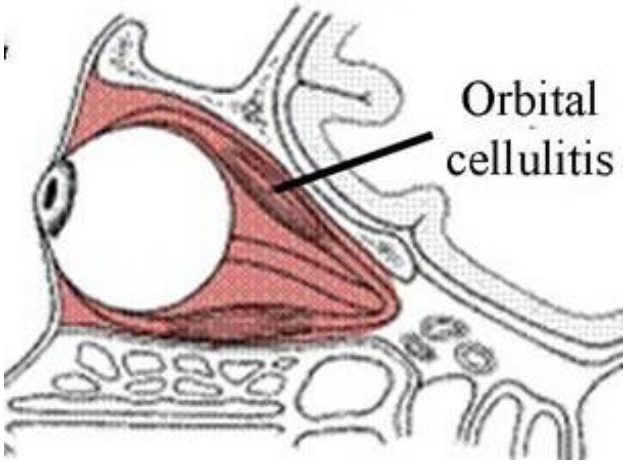
CT reveals diffuse thickening of lid and conjunctiva



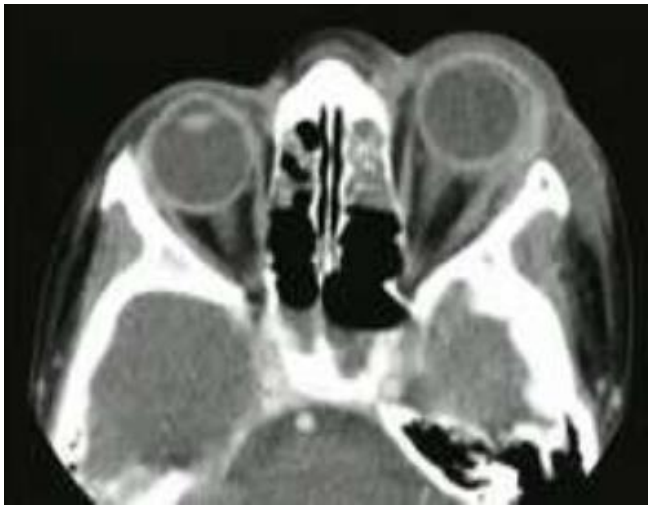
Pic. 44. CT of Preseptal cellulites

Stage II, Orbital Cellulitis/Post-septal Cellulitis

In contrast to pre-septal cellulitis, orbital cellulitis is the edema and inflammation of the orbital contents behind the orbital septum without abscess formation. Main complaints are proptosis, restricted and painful eye movements, and chemosis. Examination of the vision is of great importance as the visual loss may accompany. Some children initially may lose the ability to distinguish green and /or red color.

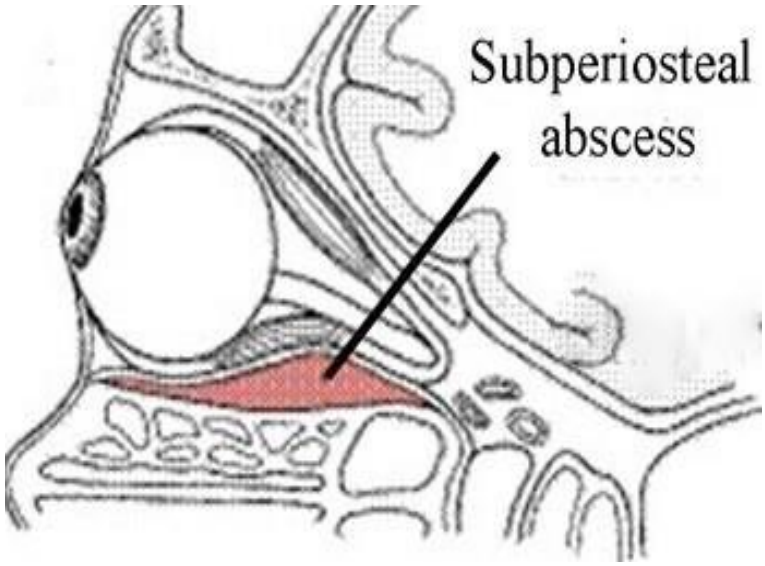


Pic. 45. Orbital cellulitis



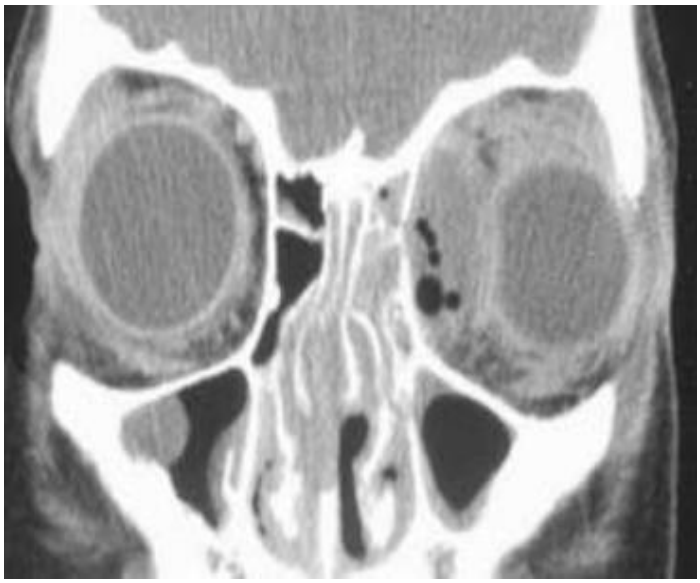
Pic. 46. CT of orbital cellulitis

Stage III, Subperiosteal Abscess



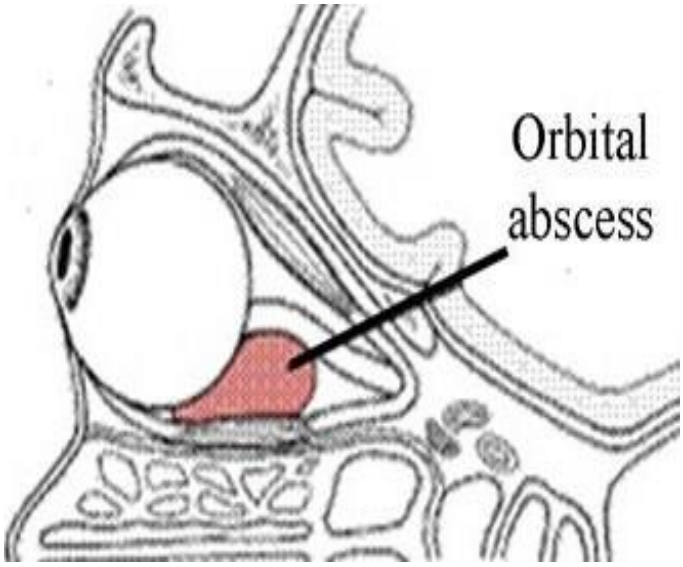
Pic. 47. Subperiosteal abscess

This condition is the formation of an abscess between the periosteum and the bone. Because of the mass effect of the abscess, the orbit is generally displaced in inferolateral direction. Proptosis, chemosis, impaired eye movements, and loss of vision are constant findings of abscess.



Pic. 48. CT of subperiosteal abscess

The CT discloses the presence of purulent collection in the medial orbital wall, between the periorbital and the orbital bone, with an extraconal location and, thus, outside the ocular muscles. The most common microorganisms are Streptococci in children and anaerobic bacteria in adults.

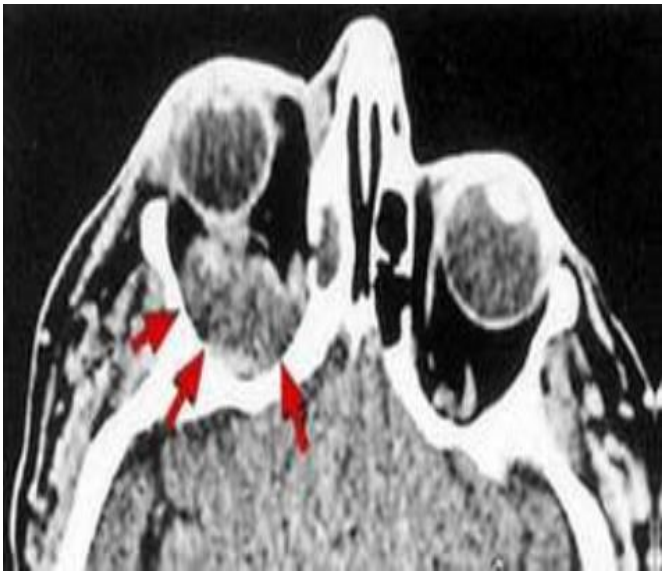


Pic. 49. Orbital abscess

Stage IV, Orbital Abscess

Orbital abscess refers to the formation of pus within the orbital content. Patients are representing complete ophthalmoplegia, severe proptosis, and loss of vision. The CT image shows purulent collection in the soft tissues around the eyeball. It may remain localized or extend through the orbital septum, emerging as a floating mass in the eyelid. It is a severe condition that can lead to amaurosis.

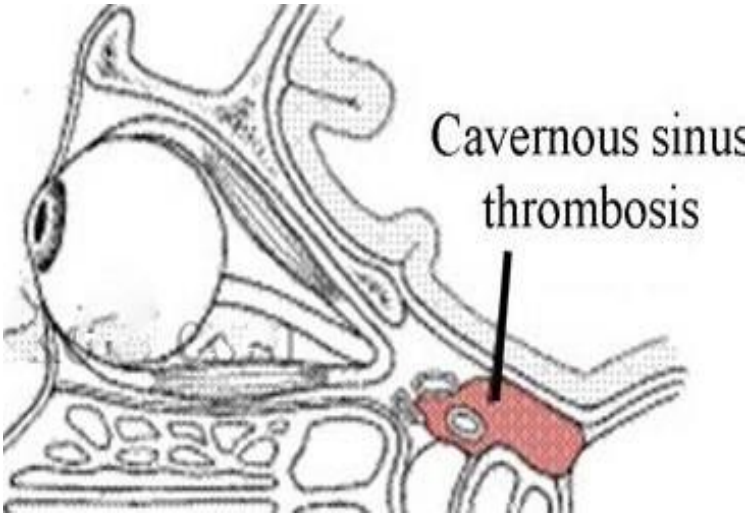
The orbital apex syndrome is a localized form of orbital cellulitis, wherein vascular-nervous lesions occur in cranial nerves III, IV, VI and in the ophthalmic branch of the V nerve, which pass through the superior orbital fissure and optic foramen. Clinically, the eyeball is fixed and pupils are dilated and nonreactive to light; ptosis, and palpebral, corneal, and conjunctival hypoesthesia are also observed.



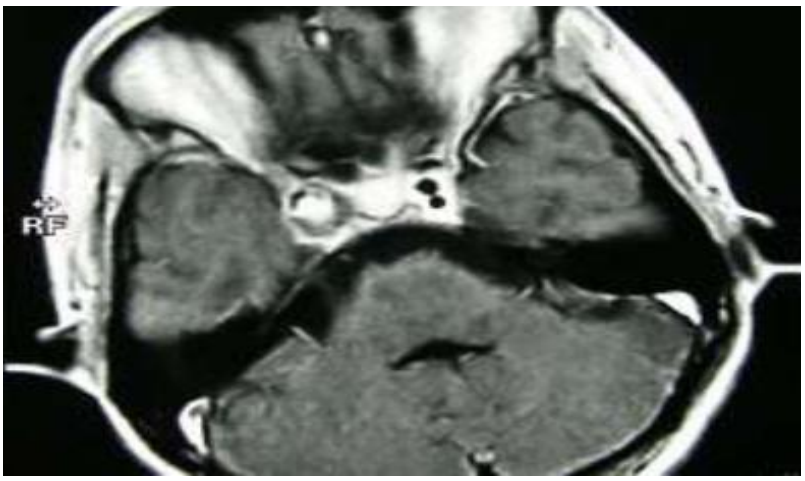
Pic. 50. MRI of orbital abscess

Stage V, Cavernous Sinus Thrombosis

This complication has the worst prognosis. Patients have bilateral ocular involvement. High fever, severe headache, photophobia, proptosis, bilateral ophthalmoplegia, loss of vision, and palsies of III, IV, V1, V2, and VIth cranial nerves are the signs of cavernous sinus thrombosis. The accompanying photophobia and neck stiffness may be mistaken for meningitis. Definitive diagnosis is made by high-resolution CT scan, which shows low enhancement compared to normal.



Pic. 51. Cavernous sinus thrombosis



Pic. 52. MRI of cavernous sinus thrombosis

Microbiology

The most frequently isolated microorganisms in orbital cellulitis and abscess are Haemophilus influenza, Streptococcus pneumoniae, S. aureus, and anaerobic bacteria (Prevotella, Porphyromonas, Fusobacterium, and Peptostreptococcus spp.)

Treatment

The outcome of medical treatment largely depends on the stage and persistence of the orbital involvement. Chandler I to III is primarily treated with antibiotics. However, if the loss of vision develops, additional surgery can be needed. Ophthalmology consultation and close monitoring of vision are essential if orbital involvement is suspected. Chandler IV and V are directly treated with urgent surgical intervention in addition to intravenous antibiotics. In the case of cavernous sinus thrombosis, another treatment agent is anticoagulants, but this is controversial since intracranial hemorrhage may occur.

Intracranial complications generally occur as a result of ethmoidal or frontal sinusitis. Infections can spread to endocranial structures by two routes, most commonly by diploic veins or rarely by eroding the sinus bones.

Non-specific symptoms such as high fever, nausea/vomiting, mental state problems, meningeal irritation signs, and frontal or retro-orbital headache are the main clinical findings. A patient with intracranial abscess can be completely asymptomatic or may show focal neurological findings such as mild affective or behavioral changes, altered consciousness, and imbalance due to increased intracranial pressure.

Cerebritis is the first stage of all intracranial complications. Then, an abscess formation occurs due to the encapsulation of necrosis in brain tissue. Most common isolated bacteria from patients with intracranial complications are anaerobic or mixed aerobic-anaerobic microorganisms. For an accurate diagnosis, generally, a CT scan is enough; MRI is indicated if sinus thrombosis is suspected. If there is a clinical suspect of meningitis, lumbar puncture is indicated after exclusion of intracranial abscess.

Intracranial complications of sinusitis include epidural and subdural empyema, sagittal sinus and cortical vein thrombosis, brain abscess, meningitis, facial osteomyelitis, and mucocele.

Pathogenesis

Intracranial extension of sinus infections can be due to retrograde thrombophlebitis, hematogenous spread, or direct bone erosion.

Bone erosion occurs as a result of osteitis (i.e., posterior wall of the frontal sinus in frontal sinusitis). Blood vessel coursing along the dura mater facilitates the bacterial penetration and a granulation tissue develops due to the inflammatory reaction of the dura resulting subdural empyema.

The second route for the spread is the diploic veins between paranasal sinuses and intracranial venous structures. This path is usually seen in acute sinusitis or acute exacerbations of chronic sinusitis. The absence of valves in these veins allows thrombosis progressing to venous sinuses, subdural and cerebral veins (retrograde thrombophlebitis).

The third route of spread is the hematogenous way which is important in the development mechanism of intraparenchymal brain abscess.

Routes for the spread of infections from sinuses to intracranial structures:

1.Venous spread; septic thrombophlebitis in venous structures of paranasal sinuses passes through valveless Breschet veins in the frontal bone and leads retrograde thrombophlebitis of meningeal veins.

2. Arterial spread; this way is the extension of the infection by direct arterial embolization.

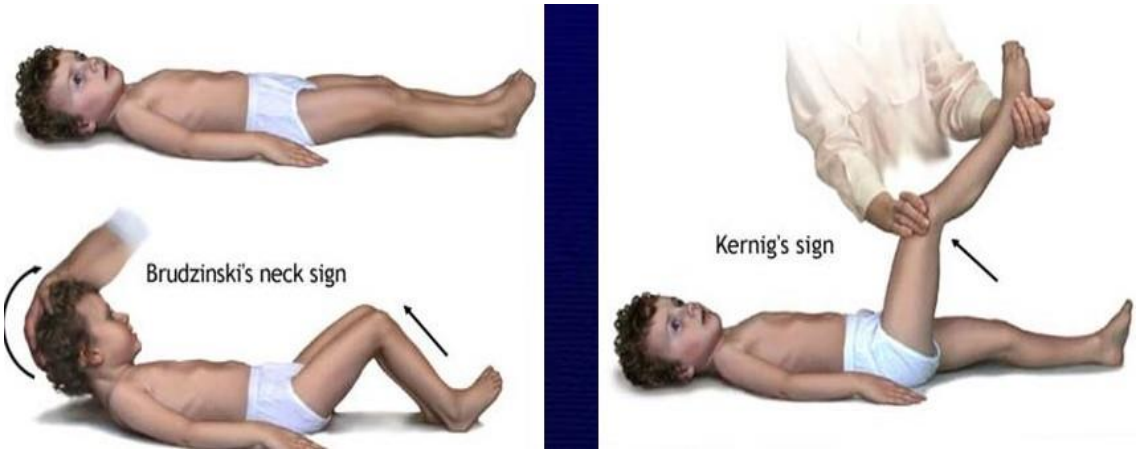
3. Bony erosion; this route is due to the bone erosion separating paranasal sinuses and intracranial structures by osteitis.

Microbiology

Anaerobes are Prevotella spp. and Porphyromonas spp., Fusobacterium spp., and Peptostreptococcus spp., aerobes are S. aureus, H. influenzae.

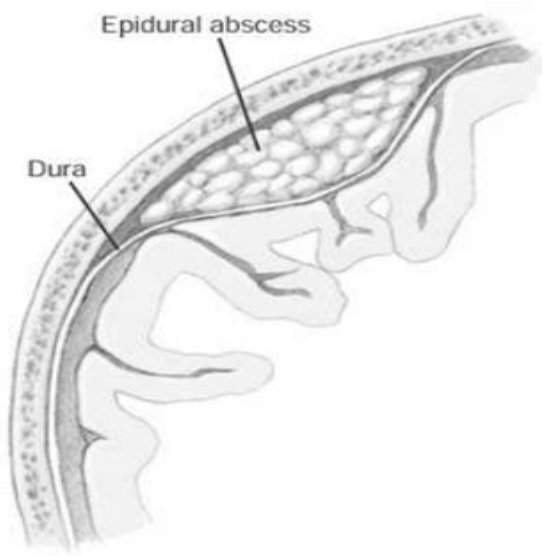
Meningitis

Clinical manifestations include fever, severe headache, neck stiffness, irritability and behavioral disorders. CT defines and delimits the disease and can identify the presence of additional complications. Lumbar puncture reveals increased proteins and cells. Lumbar puncture is contraindicated in the presence of intracranial hypertension or abscess.



Pic. 53. Meningeal symptoms

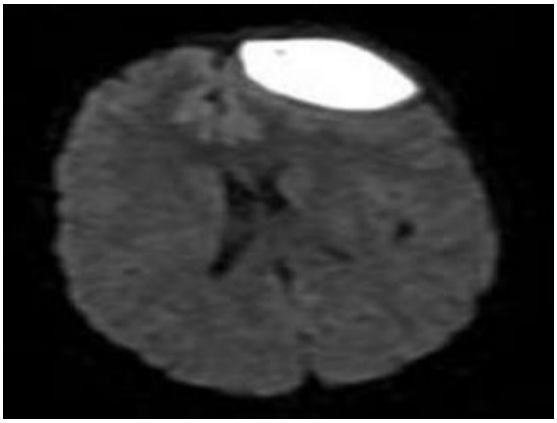
Extradural/epidural abscess



This consists of a purulent collection between the dura mater and the cranium. Occasionally, it is associated with frontal osteomyelitis. The clinical manifestations are vague, with few or no neurological signs, which, when present, include persistent headache, fever and behavioral changes.

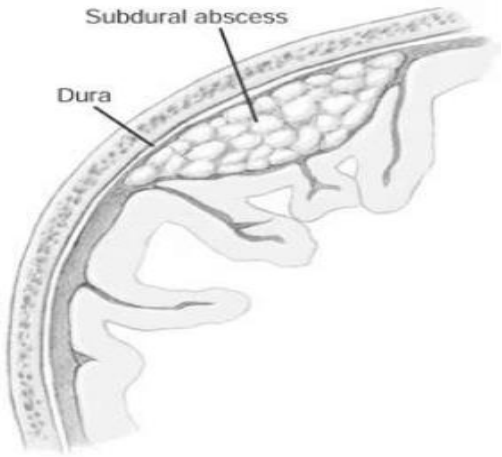
Pic. 54. Epidural abscess

The diagnosis is usually delayed because of a failure to recognize the significance of the clinical findings.

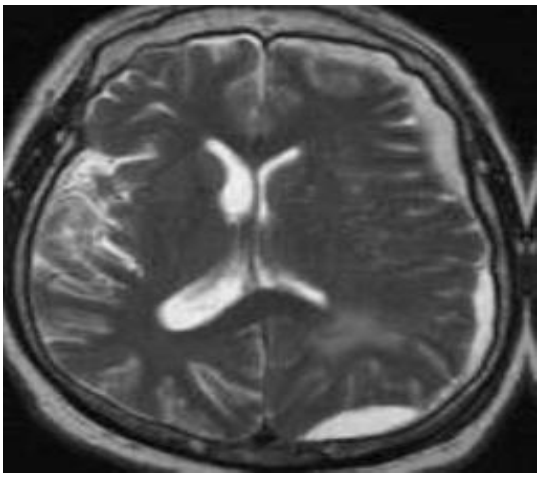


Pic. 55. CT of epidural abscess

Subdural abscess



Pic. 56. Subdural Abscess



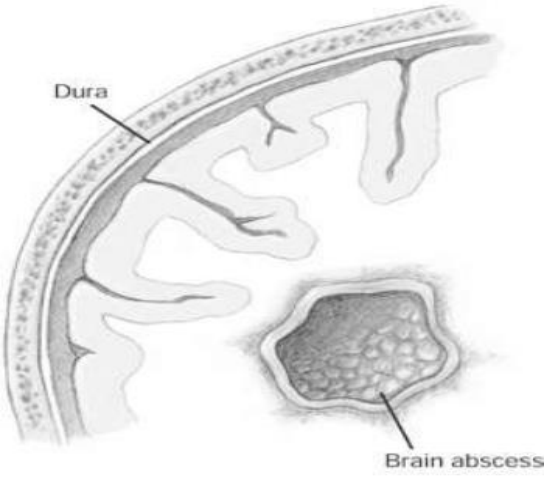
Pic. 57. Radiology of subdural abscess

Subdural abscess is characterized by the presence of purulent collection between the dura mater and the pia-arachnoid. Symptoms are severe headache, fever, decreased level of consciousness.

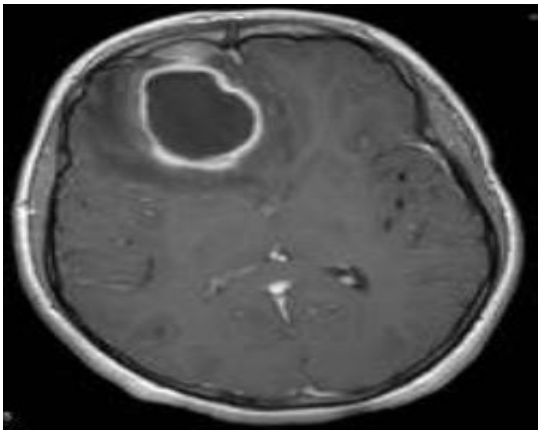
CT shows a decreasing image, not extending beyond the midline, thus differentiating from the extradural abscess.

Brain abscess

The most common location is the frontal lobe. Focal symptoms and increased intracranial pressure appear late with poor general condition, coma and cranial nerve palsy. The frontal lobe is an area of clinical silence that yields inconstant symptoms. Fever, seizures, waking period disorders, coma, motor deficit, sensory disturbances and altered vision may occur.



Pic. 58. Brain abscess



Pic. 59. Radiology of brain abscess

Imaging studies show a rounded lesion with a hypodense center and peripheral enhancement that initially is irregular but becomes more well defined as the necrotic portion progresses. Lumbar puncture is contraindicated due to the risk of brain stem decompression and herniation. Once an abscess is formed, surgical drainage is indicated combioned with paranasal sinus drainage.

Osseous Complications

Another complication of sinus infections is the osteitis of the surrounding bone tissue leading involvement of the brain and nervous system. Mucosal infection can cross the membrane and induce an inflammatory response in the underlying bone. Infection in any sinus can cause osteitis but the most common localization is frontal sinus. Typically, maxillary osteitis is common in infants. Frontal sinusitis leads to vascular necrosis of the frontal bone; osteitis occurs in the anterior and posterior table of the frontal sinus. Involvement of the anterior wall clinically presents as a “pulpy” mass under the skin of the frontal region called “Pott’s puffy tumor.” Osteitis of the posterior wall leads to meningitis, peridural abscess or brain abscess.

Abbreviations

ABRS — acute bacterial rhinosinusitis,
ACE — angiotensin-converting-enzyme,
AR — allergic rhinitis,
ARS — acute rhinosinusitis,
CN — cranial nerve,
CRS — chronic rhinosinusitis,
CRSsNP — chronic rhinosinusitis without nasal polyps,
CRSwNP — chronic rhinosinusitis with nasal polyps,
CSF — cerebrospinal fluid,
CT — computed tomography,
DAC — dentoalveolar complex,
ECA — external carotid artery,
HIV — human immunodeficiency virus,
ICA — internal carotid artery,
IFN — interferon,IL — interleukin,
IG — immunoglobulin,
INV — internal nasal valve,
LLC — lower lateral cartilages,
LLSAN — levator labii superioris alaeque nasi,
LTRA — leukotriene receptor antagonist,
MRI — magnetic resonance imaging,
MSFs — maxillary sinus fractures,
MVA's — motor vehicle accidents,
NOE — naso-orbitoethmoidal,
NP — nasal polyps,
NSA — nasal septal abscess,
NSAIDs — nonsteroidal anti-inflammatory drugs,
OMC — ostiomeatal complex,
ORIF — open reduction and internal fixation,
PSAA — posterior superior alveolar artery,
QOL — quality of life,
SMAS — subcutaneous muscular aponeurotic system,
Spp — speciales,
TESPAL — transnasal endoscopic sphenopalatine artery ligation,
TGF — transforming growth factor,

TNF — tumor necrosis factor,
ULC — upper lateral cartilages,
VRS — viral rhinosinusitis,
ZMC — zygomaticomaxillary complex.

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MCQ

1. The cribriform plate forms the roof of the nasal cavity. Which cranial bone does it belong to?
    1. Temporal bone
    2. Frontal bone
    3. Ethmoidal bone
    4. Sphenoidal bone
2. The most common site of leak in CSF rhinorrhoea is:
    1. Sphenoid sinus
    2. Frontal sinus
    3. Cribriform plate
    4. Tegmen tympani
3. In acute sinusitis the predominant organisms are except one
    1. Streptococcus pneumonia
    2. Staphylococcus aureus
    3. Hemophilus influenza
    4. Moraxella catarrhalis
4 Throught which paranasal sinus can the pituitary gland be accessed surgically:
    1. Maxillary sinus
    2. Ethmoidal sinus
    3. Sphenoid sinus
    4. Frontal sinus
5. The nasal septum is formed by all the following except:
    1. Perpendicular plate
    2. Vomer
    3. Septal cartilage
    4. Hard palate
6. Thrombophlebitis of cavernous sinus is mostly developed in:
    1. Sphenoiditis
    2. Ethmoiditis
    3. Nasal trauma and bleeding
    4. Furuncle of the nose

7. Allergic rhinitis is characterized by all except:
    1. Rhinorrhea
    2. Sneezing
    3. Pus in the middle nasal meatus
    4. Nasal obstruction
8. Which of the following sinuses drains into superior nasal meatus:
    1. Maxillary sinus
    2. Frontal sinus
    3. Sphenoidal sinus
    4. Anterior ethmoidal sinus
9. Nasal septum abscess may lead to except:
    1. Thrombophlebitis of cavernous sinus
    2. Nasal deformity
    3. Nasal obstruction
    4. Nasal septum haematoma
10. Most common bacteria leading to chronic rhinosinusitis:
    1. Streptococcus pneumonia
    2. Hemophilus influenza
    3. Moraxella catarrhalis
    4. Staphylococcus species
11. Orbital apex syndrome involves:
    1. Olfactory nerve
    2. Ophthalmic division of trigeminal nerve
    3. Maxillary division of trigeminal nerve
    4. Mandibular division of trigeminal nerve
12. All are true regarding mucocele of frontal sinus except:
    1. Cystic tender swelling
    2. Egg shell crackling can be elicited
    3. Displaces the eyeball downward and laterally
    4. Treatment is frontoethmoidectomy
13. Confirmation of the CSF from the nose can be done by except:
    1. Performing the double halo test or ring test

2. Sending samples of the fluid to the laboratory for evaluation of chloride and glucose
3. Sending samples of the fluid to the laboratory for evaluation of β2-transferrin
4. Sending samples of the fluid to the laboratory for evaluation of proteins

14. Orbital complications except:
    1. Orbital cellulitis
    2. Orbital abscess
    3. Orbital foreign body
    4. Cavernous sinus thrombosis
15. Which one of the following arteries belongs to internal carotid system:
    1. Nasaopalatine
    2. Greater palatine
    3. Sphenopalatine
    4. Anterior ethmoidal
16. Local factors of nasal bleeding except:
    1. Inflammation
    2. Injuries of the nose
    3. Heart failure
    4. Anatomical deformity
17. Systemic factors of nasal bleeding except:
    1. Colds
    2. Heart failure
    3. Deficiency of vitamin K or C
    4. Anatomical deformity of the nose
18. Which one of the following medications belongs to Anti-leukotrienes:
    1. Mometasone
    2. Montelukast
    3. Azelastine
    4. Fexofenadine

19. Which one of the following symptomps does not belong to meningeal symptoms:
    1. Kernig’s sign
    2. Brudzinski’s sign
    3. Neck stiffness
    4. Schwartz’s sign
20. Which one of the following symptomps does not belong to mild severity of allergic rhinitis:
    1. Normal sleep
    2. Normal work and school
    3. Troublesome symptoms
    4. No impairment of daily activities

Responses for MCQ

1. 3
2. 3
3. 2
4. 3
5. 4
6. 4
7. 3
8. 3
9. 4
10. 4
11. 2
12. 1
13. 4
14. 3
15. 4
16. 3
17. 4
18. 2
19. 4
20. 3

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