

CHAPTER 26

I am Choking at Night

Prof. Ahmed S. BaHammam

A 48-year-old male patient presents to the ENT clinic with a history of loud snoring that disturbs his wife. Due to the disturbing sound, she sleeps in a separate room. Upon further questioning, the patient reports interrupted sleep at night, choking attacks during sleep, apneas as witnessed by his wife, nocturia, nocturnal palpitations, nocturnal sweating, dry mouth and throat upon awakening and un-refreshing sleep. In addition, the patient complains of daytime fatigue and excessive daytime sleepiness. He reported falling asleep at work and having a motor vehicle accident (MVA) 2 months prior due to falling asleep while driving. The patient has a history of difficult-to-control hypertension and diabetes mellitus. Physical examination reveals a body mass index (BMI) of 38 kg/m², a neck circumference of 18 inches (45.7 cm), BP of 150/110 and a pulse rate of 85 beats/min. A throat examination shows a Mallampati score of IV. The patient had previously visited an ENT surgeon, who diagnosed him clinically with snoring and obstructive sleep apnea syndrome (OSAS) and recommended upper airway surgery. He comes to your clinic for a second opinion before undergoing surgery.

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Q1. What is the prevalence of OSAS?

OSAS is defined as five or more abnormal obstructed breathing events per hour of sleep and excessive daytime sleepiness. Studies have reported the prevalence of OSAS as 4% in men and 2% in women. An OSAS without

daytime sleepiness has a much higher prevalence. Studies in Saudi Arabia using the Berlin Questionnaire, which is a validated questionnaire to detect the risk of OSAS, have reported that 33.3% of middle-aged Saudi men and 39% of middle-aged Saudi women are at a high risk for OSAS.

Q2. What are the complications of OSAS?

The OSAS can lead to excessive daytime sleepiness, which increases the risk of MVAs and other accidents. In addition, there is a strong link between OSAS and cardiovascular and cerebrovascular complications such as hypertension, ischemic heart disease, arrhythmias, heart failure, pulmonary hypertension and stroke. New studies have linked OSAS with insulin resistance. In addition, OSAS can cause depression and decreased memory and concentration.

Q3. What is the pathophysiology of OSAS?

The upper airway tends to narrow during sleep, resulting in recurrent closures that cause repeated apneas despite continued efforts to breathe. These apneas result in intermittent hypoxemia and frequent arousals. Intermittent hypoxemia and arousals cause increased sympathetic activity, which increases the risk of cardiovascular complications and arrhythmias. Frequent apneas during sleep disturb sleep architecture, which results in poor sleep quality and daytime sleepiness.

Q4. What are the co-morbidities of OSA?

Several co-morbidities have been associated with OSA, including the following:

- Gender: OSA is more prevalent in men

- Age: OSA is more prevalent in old people, and the risk in women increases significantly after menopause
- Obesity
- Increased neck circumference (>17 inches for men and >16 inches for women)
- Craniofacial abnormalities affecting the jaw size: retro- and micrognathia
- Enlarged tonsils and adenoids, particularly in children

Q5. What will be the next step in managing this patient?

The American Academy of Sleep Medicine (AASM) considers polysomnography (PSG) to be routinely indicated as “*Standard*” for the diagnosis of sleep-disordered breathing.

Q6. What is PSG?

The PSG is an attended sleep study that is usually performed overnight by monitoring the neuro-cardio-respiratory physiological parameters during sleep (Figure 26.1). The PSG allows the determination of total sleep time, sleep stages, arousals, snoring, apneas, hypopneas and desaturations (Figures 26.2 and 26.3). It allows the physician to distinguish between obstructive and central sleep apnea. Each 30 seconds of a sleep study appears in a separate page and is called an “*epoch*” (Figure 26.4). A classical sleep study of 7 hours comprises approximately 800 pages. Sleep studies are scored manually epoch by epoch.



Figure 26.1: A sleep study room where the patient sleeps while the sleep technologist records the neuro-cardio-respiratory parameters.

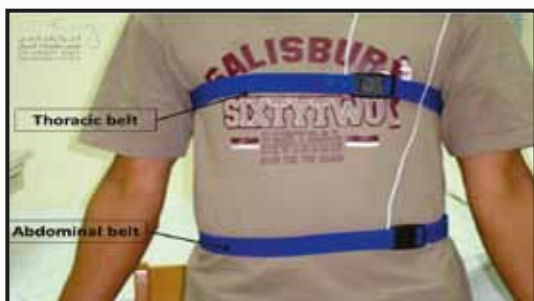


Figure 26.2: Respiratory monitoring during polysomnography; abdominal and thoracic belts monitor thoraco-abdominal breathing.



Figure 26.3: Respiratory monitoring during polysomnography; thermistor and nasal cannula monitor airflow.

Q7. What are the definitions of apnea and hypopnea?

Apnea is defined as complete cessation (90% or more reduction of nasal or oral airflow) of breathing for a duration of 10 seconds or greater. In contrast, hypopnea is defined as at least a 30% drop in nasal airflow followed by at least a 4% drop in SaO_2 .

Q8. How do we determine the severity of OSA?

The severity of OSA is determined by the number of apneas and hypopneas per hour of sleep. The index is called the apnea-hypopnea index (AHI). An AHI <5 /hour is normal; 5-15/hour is mild; 15-30/hour is moderate; and >30 /hour is severe. Other parameters that may indicate the severity of OSA include the following: the desaturation index (the number of desaturations with a 4% drop in O_2 compared with the baseline/hour of sleep) and the time spent with an O_2 saturation less than 95% or 90%.

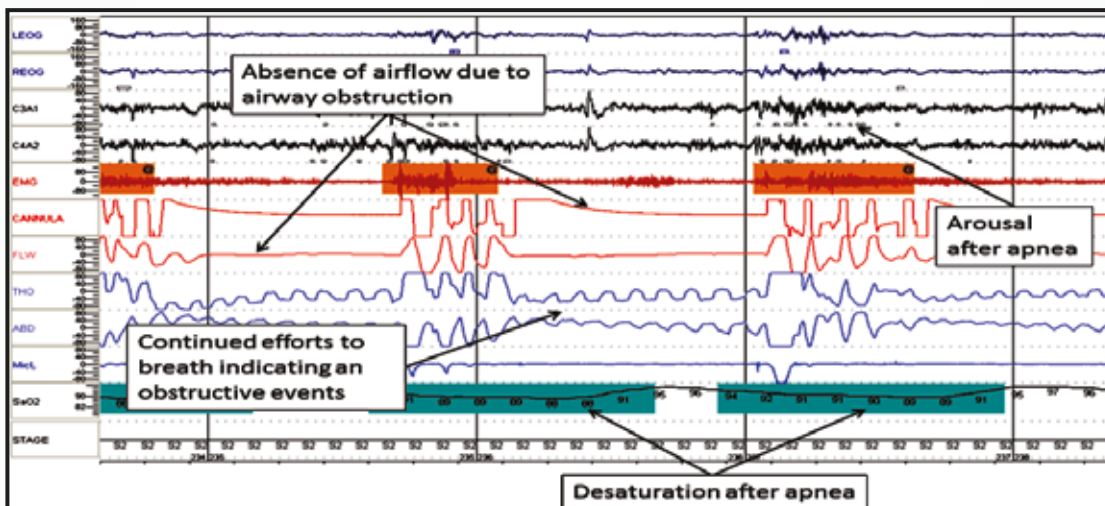


Figure 26.4: An example of a polysomnographic study of a patient with obstructive sleep apnea. LEOG and REOG: left and right electro-oculogram; C3A1 and C4A2: EEG channels; EMG: chin electromyogram; Cannula and Flow: measurements of airflow; THO and ABD: thoracic and abdominal movements; SaO_2 : oxygen saturation.

Q9. How is OSA treated?

The main component of OSA treatment is positive airway pressure (PAP) therapy applied non-invasively via an interfacing mask in the form of continuous positive airway pressure (CPAP) or bi-level positive airway pressure (BPAP). The AASM considers PAP therapy is the treatment of choice for mild, moderate and severe OSA. The PAP therapy results in significant improvement in the patient's symptoms; and several studies have shown that PAP therapy reduces the cardiovascular complications of OSA. The PAP therapy's side effects are usually minor and reversible. However, adherence to PAP therapy remains a major obstacle. Therefore, patients should receive a systematic educational program to improve adherence, and PAP usage should be monitored objectively.

Q10. How do you determine the therapeutic level of PAP?

Ideally, CPAP titration should be performed manually under PSG monitoring (Figure 26.5). The best-fitting mask that is accepted by the patient should be used. Under PSG monitoring, the CPAP pressure is increased gradually following a written protocol until snoring, arousals and all obstructive respiratory events are eliminated.



Figure 26.5: CPAP titration under polysomnographic monitoring.

Other treatment modalities have been proposed for OSA patients; however, the evidence supporting their efficacy is not as clear as PAP therapy. In this section, we summarize the most important modalities:

- *Mandibular advancement devices (MADs):*

MADs are designed by specialized orthodontists to reposition the jaw forward and downward during sleep to widen the upper airway and improve its patency. Current evidence supports the use of MADs in patients with mild-to-moderate OSA who do not tolerate PAP therapy.

- *Surgery:*

Several surgical procedures have been used to treat OSA; however, the evidence supporting most of these modalities is weak.

- *Tonsillectomy and adenoidectomy:*

Adenotonsillectomy is recommended to treat OSA in children with adenotonsillar hypertrophy. In adults, tonsillectomy is indicated for the treatment of OSA in the presence of tonsillar hypertrophy.

Q11. What are the other treatment modalities for OSA?

- *Uvulopalatopharyngoplasty (UPPP):*

The UPPP aims to reduce upper airway obstruction at the oropharyngeal level by reducing soft palate redundancy. It also aims to increase the retropalatal airway by trimming the posterior and anterior faucial pillars, excising the uvula and partially removing redundant soft palate. Randomized clinical trials comparing UPPP with control or sham surgery in OSA patients are lacking. Overall, studies have reported a success rate of 40-50% after UPPP in unselected patients with mild to moderate OSA. Nevertheless, the efficacy of UPPP appears to diminish over time. The

failure of UPPP is usually attributed to multiple sites of obstruction located more caudally in the upper airway or the persistence of the retropalatal obstruction. Based on the available evidence, UPPP can only be recommended in carefully selected patients.

- *Nasal surgery:*

The rationale behind this type of surgery is that improving nasal airway patency and reducing nasal resistance may alleviate sleep-disordered breathing. Nevertheless, based on the available data, nasal surgery as a single intervention is not recommended for the treatment of OSA.





CHAPTER 27

The Joy of Perfumes!

Dr. Rashid Al Abri

A 42-year-old female presents with a history of a diminished sense of smell for 6 months, which started after a severe URTI. The patient feels bad because she misses the pleasure of smelling perfumes. She also reports that she has also noticed a decrease in her taste sensation along with the loss of smell. She has a history of nasal trauma 2 years prior when she fell on her face at home. She denies any history of nasal obstruction, facial pain or asthma. She is a housewife, is not taking any medications, has not been exposed to harmful chemicals and has never smoked or consumed alcohol.

After the application of topical decongestants and a local anesthetic, an endoscopic examination of her nose reveals a patent nasal airway with a mildly deviated nasal septum. There are no polyps, discharge or mass lesions within the nasal cavity or in the post-nasal space.

* * * * *

Q1. What is the most likely diagnosis?

The most likely diagnosis in this patient is post-viral olfactory sensorineural loss of smell or anosmia.

Q2. Define hyposmia and anosmia.

Hyposmia is a partial loss of the sense of smell, while anosmia is the inability to perceive smell. Loss of smell may occur as a sensorineural loss following degenerative changes in the olfactory

neuronal receptors or the neuro-epithelium in the olfactory cleft. It may also occur as a conductive loss following obstruction or inflammation within the nasal vault that prevents the access of odorants to the olfactory receptors.

Q3. What are the anatomic subunits of the external nose?

See (Figure 27.1)

- Nasal dorsum

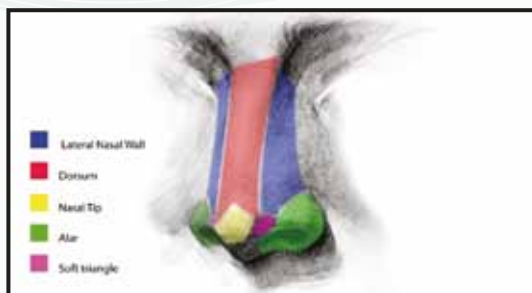


Figure 27.1: Nasal subunits

Q4. What is the nasal valve, and what its importance?

The nasal valve is the angle between the caudal quadrangular cartilage and the distal upper lateral cartilages. This area is the narrowest segment in the entire airway.

Q5. Name the nasal turbinates (conchae).

Three turbinates are found in each lateral nasal wall (Figure 27.2):

- Superior turbinate
- Middle turbinate
- Inferior turbinate

The space below each concha is called the meatus.

Q6. Describe what you mean by paranasal sinuses.

The paranasal sinuses are cavities found within the maxilla, frontal, sphenoid and ethmoid bones (Figure 27.3). They are lined with mucoperiosteum and filled with air.

Q7. Name the paranasal sinuses.

- Maxillary sinuses
- Ethmoid sinuses

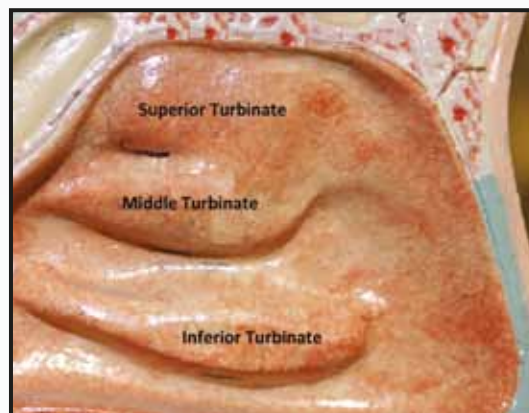


Figure 27.2: Three turbinates are found in each lateral nasal wall.

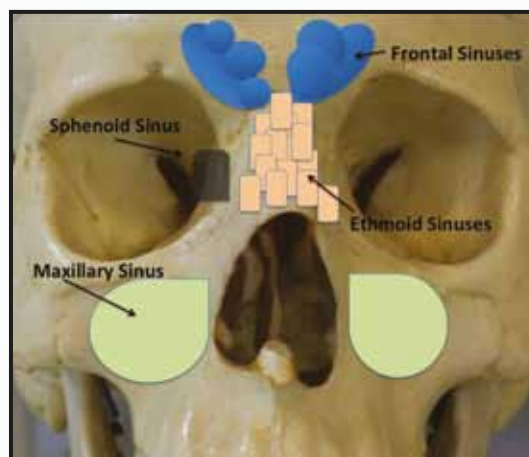


Figure 27.3: The four nasal sinuses in each side of the face.

- Frontal sinuses
- Sphenoid sinuses

Q8. Where do the paranasal sinuses drain in the nose?

- Maxillary sinuses in the middle meatus
- Anterior ethmoid sinuses in the middle meatus
- Posterior ethmoid sinuses in the superior meatus
- Frontal sinuses in the middle meatus
- Sphenoid sinus in the sphenoethmoidal recess

Q9. What is the osteomeatal complex (OMC)?

The OMC is a functional concept. It includes the infundibulum, the uncinate process, the middle meatus, the hiatus

semi-lunaris and the maxillary sinus ostium. It is the final common pathway for drainage and ventilation of the frontal, maxillary and anterior ethmoid cells.



CHAPTER 28

I Have A Runny Nose

Dr. Mohammad Aloulah

A 49-year-old female enjoyed an excellent health throughout her childhood and adult life apart from allergic rhinitis and bronchial asthma. Approximately 1 week prior, she developed nasal discharge that was yellowish in color, continuous and sometimes bloody. It was associated with fever of 38.5°C, post-nasal drip, nasal obstruction, facial pain that increases with head movement, headache and acute loss of smell. She has no history of head trauma. She has never smoked cigarettes. Her surgical history and family history were unremarkable.

Anterior rhinoscopy is performed before and after decongestion; and it reveals mucopurulent discharge in the middle meati with thick yellow-to-green post-nasal discharge. The nasal mucosa is hyperemic and edematous.

A CT scan demonstrates diffuse mucoperiosteal thickening involving the sphenoid, ethmoid and maxillary sinuses bilaterally. A clinical diagnosis of acute sinusitis is made. The patient is given a prescription for amoxicillin 500 mg orally, three times a day for 14 days. The patient is also told to dissolve one teaspoon of salt in a glass of water and to rinse both nasal cavities four times a day. She is told to pour this solution into her nares and blow it out. She is instructed not to return if the symptoms abate.

* * * * *

Q1. What are the common symptoms of nasal disease?

- Nasal airway obstruction
- Rhinorrhea (runny nose)
- Change in the sense of smell
- Facial pain or pressure
- Others, e.g., bleeding, sneezing, itching and deformity

Q2. How do we examine the nose?

Prepare the patient for the nasal examination by explaining the procedure to the patient in simple terms. The patient should be sitting up straight with his or her head at eye level and angled toward the endoscopist.

The nose is examined in three stages:

1. Examination of the external nose:
 - Inspect for congenital deformities, clefts, sinuses, deformities, shape, swellings (inflammatory, cysts and tumors) and ulcerations (trauma, neoplastic, infective).
 - Palpate for tenderness, crepitus and deformities in selected cases.
2. Anterior rhinoscopy:
 - Examine the vestibule (skin-lined part of the nasal cavity).
 - Examine the nasal cavity proper using a nasal speculum (Figure 28.1).
 - Examine before and after vaso-constriction.
3. Posterior rhinoscopy:
 - Examine the post-nasal space (nasopharynx).



Figure 28.1: A nasal speculum.

Q3. How can we examine the nasopharynx?

We can examine the nasopharynx using the following:

- Rigid naso-pharyngoscopy (Figure 28.2)
- Flexible naso-pharyngoscopy
- Mirror examination under anesthesia after palatal retraction
- Digital palpation (under anesthesia only)



Figure 28.2: A rigid scope can be useful in examination of the nasal cavity.

Q4. What is nasal endoscopy?

The nasal endoscope is a special fiberoptic telescope for the examination of the inside of the nasal cavity. It allows a very good visual examination of the nose and sinus drainage areas.

Q5. Why does the ENT specialist perform nasal endoscopy?

Nasal endoscopy offers a reliable visual view of all of the accessible areas of the sinus drainage pathways. First, the patient's nasal cavity is anesthetized; a rigid or flexible endoscope is placed in a position to view the nasal cavity. The procedure is used to detect nasal polyps or other pathologies that are not detected by routine nasal examination. During the endoscopic examination, we look for pus and take a

swab for culture as well. Moreover, the patient can see the findings in a monitor, and greater understanding and cooperation can thus be achieved.

Q6. Is there any risk from nasal endoscopy?

Although it is generally a very safe and well-tolerated procedure, the most common adverse effects of endoscopy are patient discomfort/pain, nasal bleeding and light-headedness from anxiety. Occasionally, very sensitive patients may experience a vasovagal episode and faint.

Q7. What are the advantages and disadvantages of the flexible nasal endoscope?

The fiber-optic endoscope (Figure 28.3) has the advantage of being flexible and is generally smaller in diameter, which means that it is readily manipulated in multiple directions to permit visualization of difficult areas. However, flexible endoscopy requires two hands for manipulation of the instrument and is a more difficult procedure. Traditionally, flexible endoscopy has provided lower quality visualization, but this drawback has been overcome with the development of digital flexible endoscopes and camera-at-the-tip flexible scopes.



Figure 28.3: A flexible nasal endoscope.

Q8. What are the advantages and disadvantages of the rigid nasal endoscope?

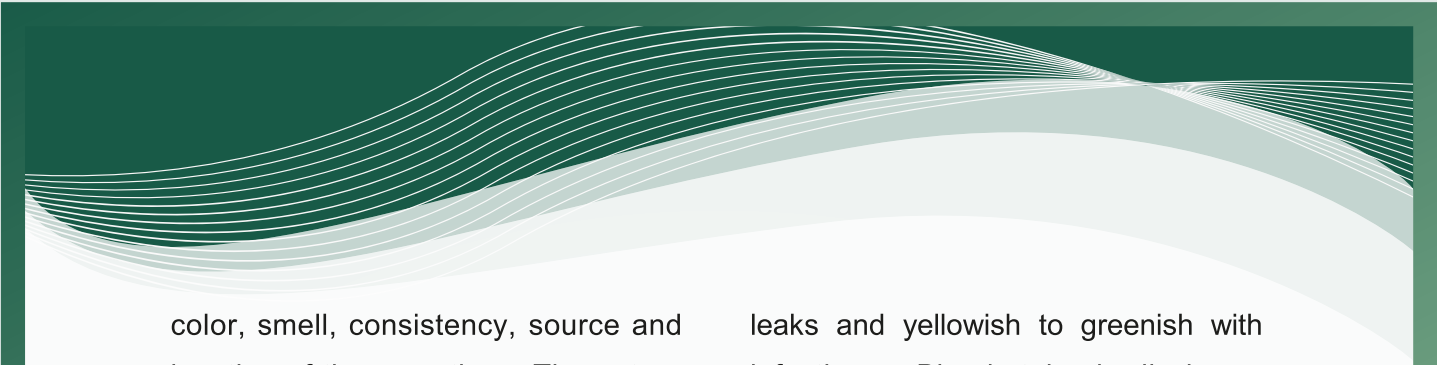
The rigid endoscope provides superior image clarity, allows the endoscopist to have a free hand to facilitate culture and tissue sampling. It is ideal for procedural work such as controlling epistaxis and performing surgery. Rigid endoscopes for the nose come in different diameters (from 2 mm to 4 mm),

Q9. What do we look for during a nasal mucosa examination?

The mucosa should be examined for color, swelling, atrophy, moisture or dryness and the presence or absence of adhesions.

Q10. What are the important characteristics of nasal secretions for diagnosis?

It is important to look for the amount,



color, smell, consistency, source and location of the secretions. The nature of the secretions can be divided into serous, mucoid, purulent, bloody and mixed. Generally, the secretions are clear with allergic reactions or CSF

leaks and yellowish to greenish with infections. Blood-stained discharge may indicate a foreign body or malignancy. Foul-smelling discharge could be due to atrophic rhinitis or an organic foreign body.

CHAPTER 29

Headache

Dr. Saad M.S. Asiri

A 34-year-old Saudi female presents with a history of a common cold that has lasted 14 days. She started to suffer from frontal headache 2 days prior to arrival at the clinic. She is febrile (39°C) and has mild nasal blockage and general weakness. A nasal examination reveals congested nasal mucosa with purulent nasal secretions from the middle meati (Figure 29.1).

* * * * *



Figure 29.1: A flexible nasal endoscope view showing pus in the middle meatus.

Q1. From the above clinical description, what features suggest the diagnosis of acute frontal sinusitis?

- History of common cold
- Frontal headache
- Temperature of 39°C
- Purulent nasal discharge

Q2. What are the most common bacteria in acute sinusitis?

- *Streptococcus pneumoniae*
- *Haemophilus influenza*
- *Moraxella catarrhalis*
- Anaerobes

Q3. Can the site of the pain indicate the most likely sinus involved? Give examples.

The site of the pain is not a very reliable indicator of which sinus is involved due to the phenomenon of referred pain. In addition, acute sinusitis very often involves multiple sinuses. Nevertheless, infection of certain sinuses have been reported to cause pain in particular sites.

- Maxillary sinus – upper jaws with

with radiation to the teeth

- Frontal sinus – over the frontal sinus in the forehead
- Ethmoid sinus – nasal bridge and inner canthus
- Sphenoid sinus – retro-orbital and occipital areas

The site of tenderness can occasionally be helpful in locating the affected sinus.

Q4. What are the common predisposing factors in the pathogenesis of acute bacterial sinusitis?

- Recurrent viral URTI
- Allergy, dental procedure and trauma
- Anatomical lesions obstructing the drainage of sinuses

Q5. What further important findings on examination will help determine the diagnosis?

Endoscopic nasal examination to reveal the following:

- Mucosal changes
- Anatomical lesions obstructing the

drainage of sinuses

- Nasal polyposis

Q6. Mention four important points in the history and physical examination to make a diagnosis of acute rather than chronic sinusitis?

Acute sinusitis:

- Symptoms lasting for less than 3 weeks
- Fever
- Purulent nasal discharge
- Headache

Q7. What is the role of a CT scan in the diagnosis and management of this case?

The CT scan is not indicated in simple acute sinusitis. In recurrent or chronic cases, a CT scan can help in identifying the following factors:

- The involved sinuses
- The presence of any congenital anatomical variations
- Any orbital or bony complication
- Any intracranial extensions

CHAPTER 30

Postnasal Drip

Dr. Hussain Abdulrahman

A 42-year-old male complains of an irritating cough that often worsens at night. The condition started 4 months prior and was preceded by an URTI diagnosed as flu by his family physician. The flu symptoms of fever, fatigue and runny nose improved with symptomatic treatment, but the nasal discharge became purulent. After 3 weeks, the patient was evaluated again by the family physician. He was diagnosed with acute sinusitis and prescribed amoxicillin-clavulanic acid in addition to a decongestant and sodium chloride nasal irrigation for 2 weeks. After this period, the patient was evaluated again with an X-ray of the paranasal sinuses that revealed partial opacification of the maxillary and ethmoid sinuses on both sides. The patient was referred to the ENT clinic. The patient reports feeling something like water trickling down his throat, which is associated with an irritating cough that often worsens at night and is sometimes associated with nasal blockage, sore throat and hoarseness.

The general physical examination is normal. The head and neck examination reveals mild tenderness to palpation of both maxillary sinuses. The nasal examination reveals mild congestion of the nasal mucosa, hypertrophy of both inferior turbinates and a deviated nasal septum (DNS) to the left. Nasal endoscopy reveals a right concha bullosa (Figure 30.1) and pus in both middle meati. An oropharyngeal examination reveals thick secretions with redness of the posterior pharyngeal wall. The laryngeal examination is normal.



Figure 30.1: Right-sided concha bullosa.

Q1. What is the diagnosis of this case?

Post-nasal drip secondary to sinusitis.

Q2. What is post-nasal drip?

Post-nasal drip is the presence of nasal secretions in the post-nasal space or throat, which creates the sensation of a tickle or itch in the throat. The mucus may be clear and watery or thick with a green, yellow or white discoloration.

Q3. What are the causes of post-nasal drip?

- Colds, flu or sinusitis
- Allergic rhinitis
- Irritating fumes from chemicals, perfumes, cleaning products or eating spicy food
- Drugs: contraceptive pills and anti-hypertensive medications
- Pregnancy: estrogen
- Weather changes: cold temperature or excess dryness of the air

Q4. What are the causes of post-nasal drip in this case?

In the early stage, the cause was the flu; and in the later stage, the cause was sinusitis.

Q5. What are the predisposing factors for sinusitis in this case?

- A history of flu
- The DNS to the left side
- The concha bullosa on the right

Q6. What are the other predisposing factors for chronic sinusitis?

- Anatomic abnormalities of the osteomeatal complex (e.g., bulla ethmoidalis, deviation of the uncinate process)
- Mucosal abnormalities: allergic and non-allergic rhinitis, nasal polyps and turbinate hypertrophy
- Environmental causes: smoking and pollution

Q7. What are the diagnostic criteria for rhinosinusitis?

The presence of two or more major criteria or one major criterion and two or more minor criteria.

Q8. What are the major and minor criteria to diagnosis chronic rhinosinusitis?

The symptoms should persist for 12 weeks or more.

The major criteria are as follows:

- Facial pain or pressure
- Nasal obstruction or blockage
- Nasal discharge or purulence
- Purulent or colored post-nasal discharge
- Purulence in the nasal cavity
- Hyposmia or anosmia

The minor criteria are as follows:

- Headache
- Fever in chronic sinusitis
- Halitosis
- Fatigue
- Dental pain
- Cough
- Ear pain, pressure or fullness

Q9. How do you examine a patient with post-nasal drip?

The physical examination should include the following:

- Complete head and neck examination
- External nasal examination including sinus palpation
- Anterior rhinoscopy
- Nasal endoscopy
- Oral and oropharyngeal examination
- Laryngeal examination to rule out gastroesophageal reflux disease

Q10. What are the possible findings in patients with post-nasal drip secondary to chronic rhinosinusitis?

- Nasal mucosa erythema and edema
- Purulent nasal discharge
- Hypertrophied nasal turbinate, DNS or nasal polyps
- Oropharyngeal erythema
- Purulent secretions on the posterior pharyngeal wall
- Tenderness on palpation of the maxillary sinuses

Q11. How do you investigate a patient with post-nasal drip secondary to chronic rhinosinusitis?

- A CT scan with coronal and axial views is the standard investigation for evaluating chronic rhinosinusitis and is indicated after the failure of maximal medical therapy and before surgery. In this case, there is a right concha bullosa, a DNS to the left and bilateral maxillary sinusitis (Figure 30.2).



Figure 30.2: Right-sided concha bullosa, DNS to the left side and bilateral maxillary sinusitis

Q12. How do you treat a patient with post-nasal drip secondary to chronic rhinosinusitis?

- Eradication of the infection: antibiotics supported by decongestants, topical steroids and saline nasal irrigation.
- Correction of predisposing factors: repair of the DNS by septoplasty and of the concha bullosa by endoscopic sinus surgery.

Q13. What is an adequate course of antibiotics in chronic rhinosinusitis?

The course of antibiotics should consist of 3-4 weeks of treatment, preferably culture-directed therapy. The therapeutic options include the following:

- A combination of amoxicillin and a beta-lactam antibiotic
- Clindamycin
- Macrolides
- Quinolone (e.g., moxifloxacin)



CHAPTER 31

Blindness

Brig. Gen. Dr. Saud Saleh Alsaif

A 40-year-old female with a history of post-nasal discharge and nasal obstruction for more than 2 years presents to the ER with left facial swelling, left eye proptosis, sudden loss of vision, headache, nausea and a low-grade fever. Her symptoms started 10 days ago with a mild headache and otalgia. She was treated with analgesics, but the symptoms worsened.

Upon physical examination, she is alert and oriented, and her vitals are stable. She has obvious swelling of the left side of the face; severe proptosis and chemosis of the left eye; and complete left ophthalmoplegia involving cranial nerves III, IV and VI. Fundoscopy shows a pale retina and mild papilledema. Her WBC count is $19,000/\text{mm}^3$ (normal 5,000-10,000). A CT scan suggests left orbital cellulitis with inflammatory changes in the ipsilateral ethmoid, sphenoid and maxillary sinuses but no evidence of an orbital abscess. An MRI shows similar findings with no element of cavernous sinus thrombosis or extension to the brain.

* * * * *

Q1. What is the most probable diagnosis?

Orbital cellulitis as a complication of an acute exacerbation of chronic sinusitis.

Q2. What are the definitions of acute, subacute and chronic rhinosinusitis?

Sinusitis can be divided into the following categories:

- Acute sinusitis, which is defined as symptoms lasting less than 4 weeks

- Subacute sinusitis, which is defined as symptoms lasting 4 to 12 weeks
- Chronic sinusitis, which is defined as symptoms lasting longer than 12 weeks
- Recurrent acute sinusitis, which is often defined as three or more episodes per year

Q3. What are the other orbital complications of sinusitis?

According to the Chandler criteria, they

can be classified as follows:

1. Preseptal cellulitis: The infection is restricted anteriorly by the orbital septum and presents with eyelid swelling (Figure 31.1) and pain without involvement of the orbital contents.
2. Orbital cellulitis: The infection involves the soft tissues of the orbit, causing diffuse inflammation without suppuration. It may cause ophthalmoplegia, chemosis, exophthalmos and low visual acuity.
3. Subperiosteal abscess: A collection of pus between the lamina papyracea and the periorbita in the medial wall of the orbit pushing the globe inferolaterally. There may be vision loss with proptosis and exophthalmos (Figure 31.2).
4. Orbital abscess (Figure 31.3): The formation of pus within the orbital tissue. It may cause severe exophthalmos, pain, fever and loss of vision.
5. Cavernous sinus thrombosis: The end stage of orbital infection extending to the intracranium. The classic presentation is the abrupt onset of unilateral periorbital edema, headache, photophobia and proptosis along with ptosis, chemosis and cranial nerve palsies (III, IV, V and VI). The sixth nerve palsy is the most common. Sensory deficits in the ophthalmic and maxillary branches of the fifth nerve are common. Periorbital



Figure 31.1: Eyelid swelling due to preseptal cellulitis as a complication of sinusitis. (Courtesy of Dr. Ameen Al-Herabi)

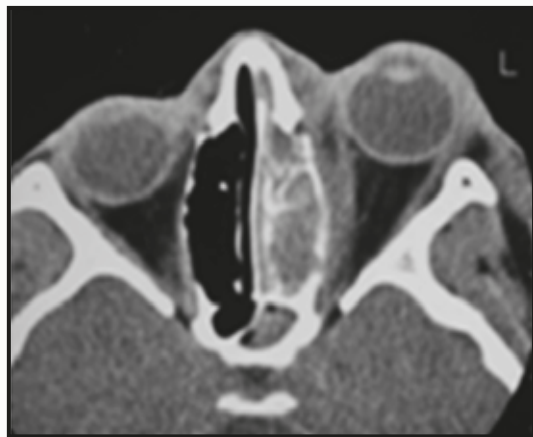


Figure 31.2: An axial CT scan showing sinusitis with intraorbital extension.



Figure 31.3: Orbital abscess.

sensory loss and an impaired corneal reflex may be noted. Papilledema, retinal hemorrhages and decreased visual acuity and blindness may occur due to venous congestion within the retina.

Fever, tachycardia and sepsis may also be present. Headache with nuchal rigidity may occur. The pupils may be dilated and sluggishly reactive. The infection can spread to the contralateral cavernous sinus within 24-48 hours of the initial presentation.

Q4. What are the other complications of sinusitis?

1. Intracranial complications:
 - Epidural abscess
 - Subdural abscess
 - Meningitis
 - Brain abscess
 - Cavernous sinus thrombosis
2. Bony complications:
 - Osteomyelitis of the frontal bone
 - Pott's puffy tumor, which may be associated in 60% of cases with other abscesses in the following locations:
 - Pericranial
 - Periorbital
 - Epidural
 - Subdural
 - Intracranial

Q5. What are the causes of loss of vision?

- Ischemic optic neuropathy
- Compressive optic neuropathy
- Inflammatory optic neuropathy

Q6. What are the main routes of infectious spread to the orbit and cranium?

A. Direct spread:

- Osteitis (compact bone)
- Osteomyelitis (cancellous bone)
- Osteoporosis (pressure atrophy due to polyps, mucocoeles or pyocoeles)
- Fracture line (congenital/accidental/iatrogenic trauma)

B. Venous spread:

- Septic venous thrombosis (in diploic veins during osteomyelitis)
- Thrombosis in the minute veins of the sinus mucosa that communicate with the diploic and dural sinuses, leading to intracranial complications
- Septicemia and pyemia

C. Lymphatic spread:

- Perivascular lymphatics convey the infection through vascular foramina to form sub-periosteal abscesses

D. Perineural spread:

Through the olfactory nerve to the subarachnoid space

Q7. What are the medical treatment for orbital complications?

- Hospital admission
- IV antibiotics
- Topical vasoconstrictors
- Anti-inflammatory agents
- ±Anticoagulants (for thrombophlebitis)
- ±Steroids

Q8. What are the indications for surgical interference in the treatment of the orbital complications?

- Abscess formation on a CT scan

- Progressive diminution of visual acuity
 - Increasing proptosis
 - No improvement after 48-72 hours of intensive medical treatment
 - Severe fungal infection in immunocompromised patients (Figure 31.4)

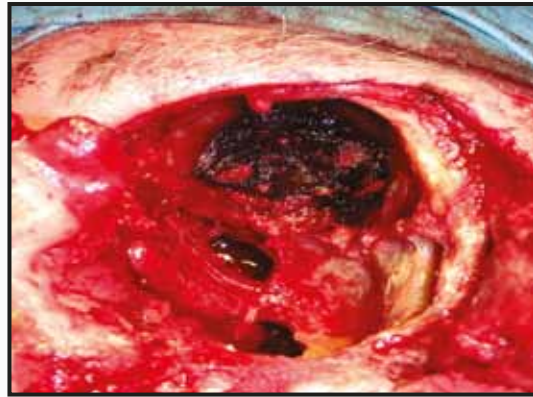


Figure 31.4: External cleaning of the left maxillary sinus and orbital exenteration were required for a severe fungal infection.

CHAPTER 32

Nasal Obstruction

Dr. Ali Alamri

A 23-year-old male presents to the Primary Care Clinic complaining of bilateral persistent nasal blockage, headache, and frequent nasal bleeding for several years. He reported no nasal trauma or nasal surgery. He was referred to the ENT clinic for a consultation. Upon examination, his vital signs are normal. Examination of the nose shows mild external nasal deformity and a moderately deviated septum toward the left with enlargement of the right inferior turbinate with poor airway bilaterally. The rest of the ENT examination is unremarkable.

* * * * *

Q1. What is the nasal septum?

The nasal septum is a thin sagittal mid-line anatomical structure that divides the nasal cavity into nearly equal halves.

More than 80% of all patients have a nasal septum not located in the mid-line, but the condition is usually asymptomatic.

Q2. What are the main arterial blood vessels of the nasal septum?

The posterior part of the septum is supplied by the sphenopalatine artery, and the inferior part is supplied by the greater palatine arteries. Both are branches of the

maxillary artery, which is a branch of the external carotid artery. The septum is anteriorly supplied by the superior labial artery branch of the facial artery, which is a branch of the external carotid artery. The superior aspect of the septum is supplied by the anterior and posterior ethmoid arteries, which are branches of the ophthalmic branch of the internal carotid artery.

Little's area lies at the most anterior part of the nasal septum, where the four arteries form a vascular anastomosis called Kiesselbach's plexus (Figure 32.1). This is the most common site of anterior epistaxis.

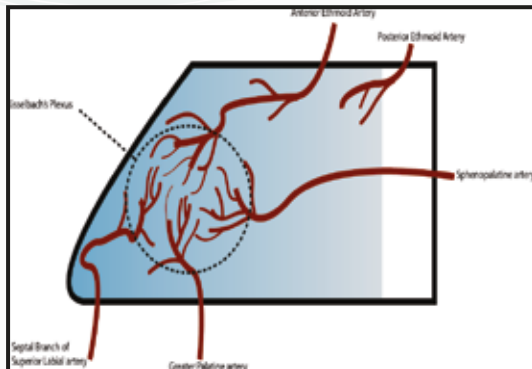


Figure 32.1: Kiesselbach's plexus

Q3. What are the functions of the nasal septum?

The septum is important to support the external nose, resist trauma, regulate airflow and support the nasal mucosa.

Q4. What are the main components of the nasal septum?

The two main components are as follows:

- A. Cephalic (posterior) portion: the bony septum, which is composed of the perpendicular plate of the ethmoid and the vomer.
- B. Caudal (anterior) portion:
 - Cartilaginous septum, formed by the quadrangular cartilage
 - Membranous septum, formed by the two layers of nasal skin
 - Columellar septum, containing the medial crurae of the alar cartilages

Q5. What is a deviated nasal septum (DNS)?

It is a septal deviation from the midline toward one nasal cavity or both, with functional and cosmetic implications.

Q6. What are the different etiologies of a DNS?

- Lateral blow to the nose, leading to displacement of cartilage from the vomerian groove and maxillary crest
- Midface trauma, a frontal blow or crushing blow leading to buckling, twisting and duplication of nasal septum
- Labor trauma leading to displacement and dislocations
- Abnormal intrauterine presentations of the fetus
- Disproportional septal nasal growth compared with palatal and skull base growth
- Cleft palate, cleft lip or dental abnormalities

Q7. What are the long-term effects of nasal septal deviations?

• **Mechanical Nasal Obstruction**

Obstruction can occur due to septal deviation to one side and hypertrophic changes in the turbinates on the other side.

• **Nasal Mucosal Changes**

Changes can occur due to air current misdirection, leading to excessive dryness, crust formation, ulceration, bleeding (crust separation) and infection.

• **Structural Changes**

Changes include hypertrophic turbinates, concha bullosa on the opposite side, elevation of the floor of the

nasal cavity, distortion of the palate and orthodontic abnormalities.

- **Neurological Changes (Sluder's Syndrome)**

Changes occur due to pressure on an adjacent sensory nerve, producing pain (e.g., the anterior ethmoid nerve).

- **Changes in the Nasal Appearance**

Depending on how severe the deviation is, the nose may deviate in the direction of the cartilage, which is called tension nose. Therefore, it is crucial to fix the septum before correcting the external shape of the nose.

Q8. What are the most common presentations of patients with a DNS?

- Nasal obstruction of one or both nostrils
- Frequent nasal bleeding
- Facial pain and headache
- Diminution of the sense of smell (hyposmia)
- External deformity

Q9. What are the most important tools required to diagnose a DNS?

- Proper history
- Physical examination, especially of the external nasal contour, nasal valve, septum and nasal cavities using the cold spatula test (placing a cold metallic tongue depressor underneath the nostril and compare the fogging produced by each side) and Cottle's test (pulling the cheek laterally to open the nose more).

- Anterior and posterior rhinoscopy before and after administering a nasal decongestant
- Nasal endoscopy

Q10. What are the treatment options for a DNS?

There is no medication to cure a DNS. The only effective solutions to the problem are as follows:

- Classical septoplasty
- Endoscopic septoplasty
- Submucous resection

Q11. What are the possible complications of DNS surgery?

- Hemorrhage
- Septal perforation (Figure 32.2)
- Septal hematoma
- Septal abscess
- Persistent nasal obstruction due to the failure of adequate resection
- Saddle nose deformity due to excessive resection
- CSF rhinorrhea
- Anosmia
- Synechia
- Toxic shock syndrome



Figure 32.2: Septal perforation can be an asymptomatic complication of septal surgery.



CHAPTER 33

Nose Sounds

Dr. Ahmed Al Arfaj

A 25-year-old male, serving a 3-month sentence in jail for drug abuse, complains of a whistling sound from his nose that occurs during breathing, following a fight with his cellmate 3 weeks ago. Immediately after the fight, he had a severe nose bleed that stopped with cold compression. However, his nose has continued to bleed mildly on and off since the fight. He also states that the nose bleed improved following the use of an ointment in the nose. He is otherwise completely healthy.

Upon physical examination, his throat, ears and neck are clear. A nasal examination reveals a defect (perforation) in the septum through which the deeper part of the other nasal cavity can be seen.

* * * * *

Q1. What is a nasal septal perforation?

A nasal septal perforation is a defect in any portion of the cartilaginous or bony septum with no overlying mucoperichondrium or mucoperiosteum on either side (Figure 33.1). A nasal septal perforation provides direct communication between the right and left nasal cavities. Septal perforation is an uncommon but bothersome illness and treatment is difficult, particularly with large perforations. Patients with septal perforations are most commonly observed both

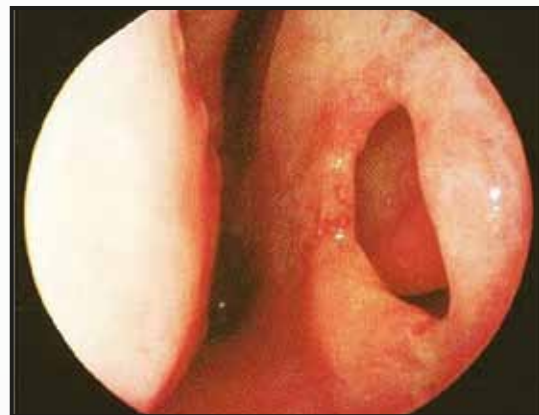


Figure 33.1: A small septal perforation

with and without symptoms after a previous nasal or sinus surgery, nose picking or cocaine use. Small perforations refer to those with a diameter

of ≤ 0.5 cm; medium perforations have a diameter ranging between 0.5 and 2 cm; large perforations have a diameter > 2 cm (Figure 33.2).

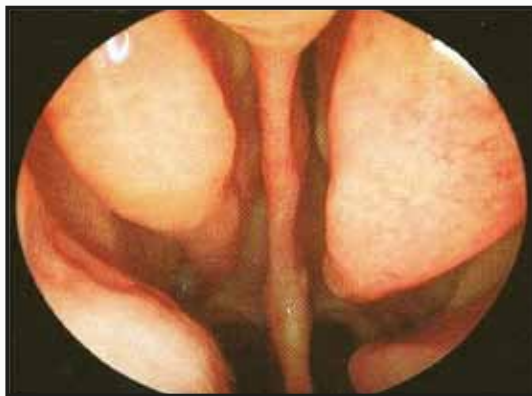


Figure 33.2: A large septal perforation down to the nasopharynx

Q2. What is the pathophysiology of septal perforation?

Nasal septal perforations result from trauma to the mucoperichondrium of the septum. A diminished blood supply can lead to cartilaginous and mucosal necrosis. After perforation occurs, the mucosal edges epithelialize, preventing closure of the defect. Symptoms arise from the altered nasal laminar airflow and may be severely disturbing to the patient (Figure 33.3). Some patients may be completely asymptomatic.



Figure 33.3: A medium-sized septal perforation may be more symptomatic than small or large perforations.

Q3. Based on your understanding of this pathophysiology, give some examples of etiologies.

The causes can be broadly classified as follows:

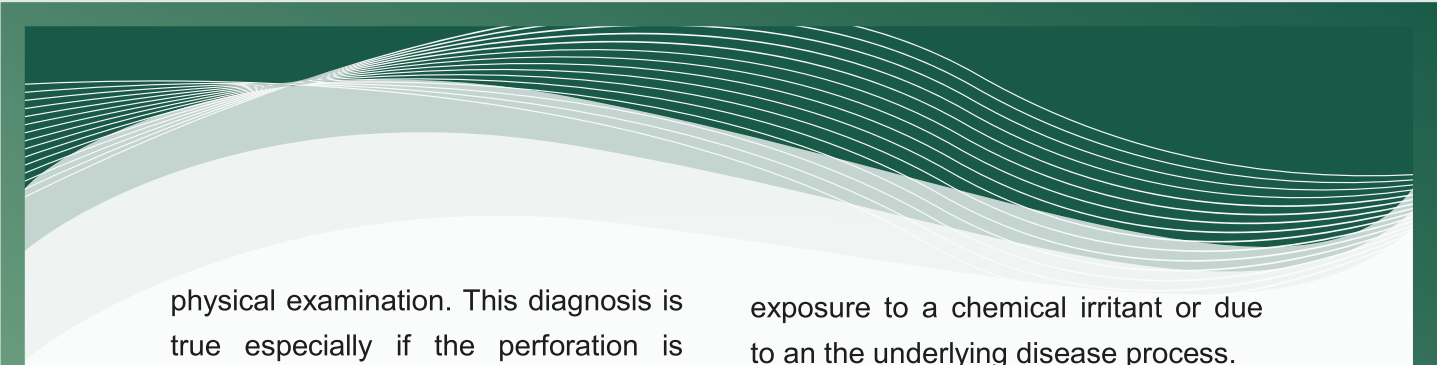
1. Trauma - external fractures and septal hematomas
2. Self-inflicted – foreign body and nose picking
3. Iatrogenic – post-surgery, e.g., septoplasty, rhinoplasty, sinus surgery, turbinate surgery, septal cauterization, packing
4. Drug use – vasoconstrictor sprays, steroid sprays, cocaine and smoking
5. Inflammatory causes – collagen vascular diseases, vasculitis, sarcoidosis, Wegener's granulomatosis and renal failure
6. Chemical irritants – acids (sulfuric, chromic, hydrochloric), chlorines, chemical and industrial dust, lime, etc.
7. Neoplastic – adenocarcinoma, squamous cell carcinoma, metastatic tumors and midline granulomas
8. Infectious – tuberculosis, syphilis, rhinoscleroma, leprosy, rhinosporidiosis and other fungal species (*Mucor* species)

Q4. What could be the pathology in such a case?

Cocaine use should be considered.

Q5. What are the symptoms and signs of septal perforation?

Septal perforation may be asymptomatic and noted only accidentally during a routine



physical examination. This diagnosis is true especially if the perforation is small and posterior. Other patients may complain of crusting and bleeding along with rhinorrhea but only rarely of pain. A symptom of pain suggests the possibility of chondritis, which can occur around the edges of the exposed cartilage at the circumference of the perforation. If the perforation is small, some patients may complain of nasal whistling. In contrast, if the perforation is large, they may complain of nasal obstruction because it disrupts the normally separate lamellar flows. Anterior perforations of the septum are more commonly symptomatic because this area of the nose is likely to be drier due to airflow. Large perforations can lead to atrophic rhinitis. Long-term large perforations may even result in a saddle nose deformity from the lack of dorsal nasal support, producing both aesthetic and functional problems.

Q6. If the perforation is anterior, what is the anatomical significance?

The nasal septum comprises the quadrangular cartilage anteriorly. This cartilage is difficult to reconstruct and has a rich blood supply, which causes frequent bleeding.

Q7. What is the natural history of septal perforation?

Most small perforations enlarge due to continuation of the inflammation as a result of the dryness of the residual nasal mucosa secondary to the turbulent air flow or as a direct effect of continued

exposure to a chemical irritant or due to an the underlying disease process.

Q8. What measures can be taken to reduce the symptoms and the progression of the disease?

If the nose is allowed to dry up internally, more crusting develops. The patients' attempts to pull out the crusts result in more bleeding, and the condition worsens. Patients should be instructed to irrigate the nose several times a day with saline and apply an ointment to reduce dryness and crusting.

Q9. What is the role of cocaine use in the etiology of septal perforation?

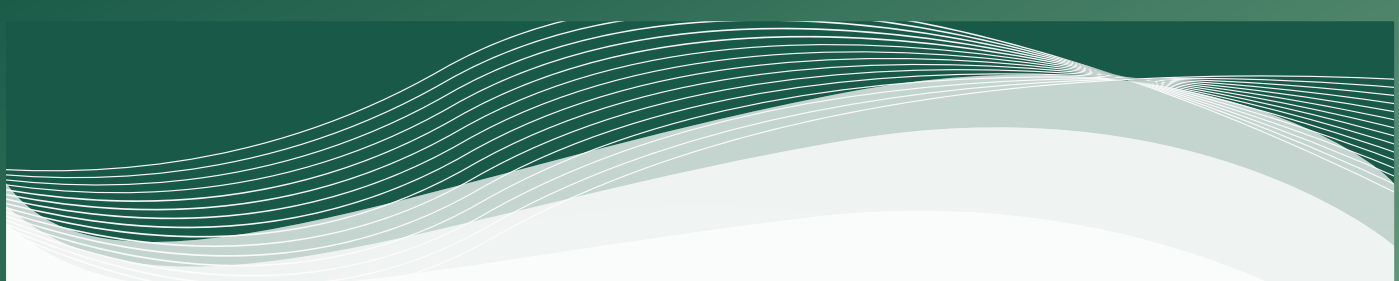
With extensive cocaine use, the normal ciliated respiratory epithelium undergoes squamous metaplasia, leading to crusting. In addition, there is a decreased vascular supply due to vasoconstriction and scarring. The quality of the remaining septal cartilage may also be compromised, leaving it thin and weak.

Q10. Why was the patient told that there was no hope for repairing this perforation surgically?

Sometimes, less-experienced physicians may tell patients (incorrectly) that such perforations are not repairable, which delays surgical treatment and may allow a small perforation to enlarge to an inoperable size.

Q11. Would you consider doing a biopsy of the perforation?

A septal perforation may be the first



sign of a potentially life-threatening general systemic process. However, biopsy is not recommended on a routine basis. It should be considered if no clear underlying cause (e.g., the presence of a mass) is found or if there is excessive bleeding or a history of cancer. The biopsy should be taken from the posterior edge of the perforation and should include enough tissue area away from the perforation to give the pathologist a representative tissue sample and not just an area of chronic inflammation.

Q12. What treatment options can you give to the patient?

The treatment options are broadly classified as medical or surgical.

Asymptomatic patients with perforations rarely require treatment and should be advised to keep the nose moist in dry climates using petroleum-based ointment. For those patients with a considerable amount of crusting, frequent therapy with nasal irrigation and ointments / emollients is indicated

Silicone buttons are ideal for patients who are not good surgical candidates for medical reasons and should also be considered in patients with a chronic or recurrent disease process and in any patient with continued cocaine use. Silicone button prostheses do not cure the perforation, but they help to restore normal nasal airflow and keep the edges

of the perforation moist. When these buttons are in place, periodic nasal irrigation may be necessary to keep the airway clean. The prosthesis may need to be removed at least annually for adequate cleaning and an integrity check.

Surgical Principles and Procedures

The main aim of surgery is to repair the perforation and restore normal nasal function and physiology. Methods that use skin grafts or oral buccal mucosal grafts may be successful, but they can leave the patient with a dry nose that continues to crust as these grafts either shed or dry out. Many types of connective tissue grafts have been used to interpose between the repaired septal flaps, including mastoid periosteum, temporalis fascia, pericranium, septal bone or cartilage, fascia lata and acellular dermal allografts. Currently, the most commonly used grafts are the temporalis fascia and the dermal and submucosal allografts.

Q13. What factors affect the outcome of surgical repair?

A successful outcome in this operation depends upon many factors, such as the cause of the disorder, the size and location of the perforation, the experience and skill of the surgeon, the medical condition of the patient and post-operative cooperation and compliance.

CHAPTER 34

Nose Bleed

Dr. Osama A. Marglani

A healthy 20-year-old male presents to the emergency department with facial trauma after a fight. The trauma occurred 2 hours ago, and the patient developed nasal bleeding that has not stopped as of the time of presentation. He also complains of a right nasal obstruction and neck pain. He is a smoker.

Physical examination reveals a conscious, Glasgow coma scale (GCS) 15 patient with stable vital signs. His nose exhibits nasal swelling and deformity to the right with swelling of the right side of the septum. He has no other injuries and no ocular or neurological compromise.

* * * * *

Q1. What is the likely diagnosis?

Nasal fracture with septal hematoma.

Q2. What imaging will you need to confirm the diagnosis?

None. There is no clear evidence for the role of radiography in the management of nasal fractures. Plain films have up to a 66% false-positive rate as a result of the misinterpretation of normal suture lines. In addition, old fractures are difficult to distinguish.

Q3. What other tests should be performed and why?

An X-ray of the cervical spine may be performed to rule out neck injuries.

Q4. What are the initial lines of management this patient needs on arrival to the ER?

- Clear his airway, breathing and circulation (ABCs).
- Insert an IV line.
- Perform a routine pre-operative work-up.

Q5. How will you manage the nose bleed?

If the bleeding is anterior and the site can be localized, then it can be cauterized using silver nitrate (Figure 34.1). Otherwise, a formal anterior packing can be inserted

(Figure 34.2). If the bleeding is posterior (from branches of the sphenopalatine), then posterior packing may be required.

Surgical methods such as direct endoscopic vessel cauterization or ligation offer better hemostasis and patient comfort. Angiography with embolization can also be considered in cases that are refractory to more conservative approaches.



Figure 34.1: Silver nitrate stick



Figure 34.2: Merocel nasal packing enlarges with fluid. The tongue depressor is included for size comparison.

Q6. Name some local causes of epistaxis.

- Trauma
- Inflammatory reactions: e.g., rhinitis, sinusitis
- Anatomic deformities: e.g., deviated nasal septum
- Foreign bodies
- Intranasal tumors
- Surgery

Q7. Name some general causes of epistaxis.

General:

1. Cardiovascular: hypertension

2. Hematological:

- Platelets (thrombocytopenia)
- Hemophilia (coagulation)
- Aspirin (anti-platelet)
- Heparin (anti-coagulant)
- Leukemia – Hodgkin's lymphoma

3. Hepatic: thrombocytopenia

4. Vasculitis

Q8. What is the most common site for bleeding? Name the arterial supply to this area.

The most common site is Little's area (an anastomosis of four arteries), which is supplied by:

- Greater palatine
- Superior labial
- Anterior ethmoid
- Sphenopalatine

Q9. Which of these arteries can be ligated endoscopically?

Sphenopalatine.

Q10. After controlling the bleeding, the septal swelling was found to be large, soft and bluish as in Figure 34.3. What is the diagnosis?

Septal hematoma.



Figure 34.3: Right side anterior nasal septum swelling.

Q11. How will you manage this and when?

Incision and drainage performed immediately.

Q12. What complications will arise from missing this diagnosis?

The complications include the following: abscess formation 3-7 days after trauma; necrosis of the septal cartilage; and long-term problems such as saddle-nose deformity, perforation and columellar retraction. Spread of septal infection may lead to orbital

infections and intracranial complications, e.g., meningitis and cavernous sinus thrombosis.

Q13. What other complications can arise from nasal packing?

- Sinusitis
- Septal hematoma/perforation
- External nasal deformity
- Mucosal pressure necrosis
- Vasovagal episode
- Balloon migration
- Aspiration



CHAPTER 35

Foul Nose Discharge

Dr. Sami Al Harethy

A 3-year-old boy is brought to the emergency room by his parents with snoring, mouth breathing and foul smelling, occasionally bloody thick nasal discharge for 6 months. The problem worsens during URTIs. He has been taken several times to a clinic near his home and is always managed as having a common cold. His examination reveals a mouth-breathing child with erythematous skin over the right nostril and a thick, mucopurulent, foul-smelling, anterior nasal discharge. Physical examination of the ear, throat and neck is normal.

* * * * *

Q1. What are the possible causes of this child's problem?

- Nasal foreign body
- Rhinosinusitis
- Unilateral choanal atresia

Most children deny putting anything in their nose. However, the parents may suspect that a foreign body is present, which should be taken seriously even if the patient is asymptomatic.

Q2. What is the most likely diagnosis?

The diagnosis is a nasal foreign body until proven otherwise. This diagnosis should be the first possibility on the differential diagnosis that should be ruled out in a child or mentally retarded patient who has unilateral foul-smelling nasal discharge.

Q4. What is the main diagnostic tool?

Physical examination is the main diagnostic tool with adequate inspection of the nasal cavity, using a headlamp and/or an endoscope. Sedation or even general anesthesia is often required in the pediatric population.

Q3. How useful is the history in these cases?

Q5. Where is the most common location for nasal foreign bodies?

Nasal foreign bodies tend to be located on the floor of the nasal passage, just below the inferior turbinate or anterior to the middle turbinate.

Q6. When are imaging studies indicated?

Imaging studies are usually indicated when another diagnosis (tumor, sinusitis) is considered or if there is a concern for an aspirated or ingested foreign body. Additionally, an X-ray might be useful if the FB is radio-opaque.

Q7. What are the symptoms of nasal foreign bodies?

Some nasal FBs (especially if small and inorganic) may be asymptomatic and discovered only during a routine examination for unrelated illness. However, the most common symptoms are unilateral nasal obstruction and discharge. If the FB is composed of organic material, then the discharge is usually foul smelling and is sometimes blood stained.

Q8. How can nasal foreign bodies be classified?

- Inorganic (e.g., beads, small parts from toys): often asymptomatic and may be discovered accidentally
- Organic (e.g., food, rubber, wood, sponge): tend to be more irritating to the nasal mucosa and thus may produce early symptoms.

Q9. What are the complications of nasal foreign bodies?

- Bleeding (most common)
- Sinusitis
- Nasal septal perforation
- Aspiration

Q10. What are rhinoliths?

Rhinoliths are long standing firmly impacted foreign bodies in the nose that are coated with calcium, magnesium, phosphate or carbonate (Figure 35.1). They are radio-opaque and typically found on the floor of the nose. They can remain undetected for years.



Figure 35.1: Endoscopic picture of rhinolith in the nasal cavity

Q11. How the management of metallic button batteries is different from other FBs?

This finding is considered as a very urgent situation and requires early intervention because it can cause severe damage within a short time. The damage occurs because of the following:

- Low-voltage electrical currents
- Electrolysis-induced release of sodium hydroxide and chlorine gas
- Liquefactive necrosis if the alkaline contents leak out (which is worse than acid)

Q12. When are nasal foreign bodies considered an emergency?

- If the foreign body has been inhaled into the person's throat and the person is choking
- If the object falls back into the throat and is swallowed.
- If the foreign body is an object that contains chemicals, such as button batteries.
- If the foreign body is made of food material.

Q13. What should be done immediately after removing a nasal foreign body?

After careful removal of the foreign body (Figure 35.2), a “second look” must be performed because there can be additional foreign bodies. Also, it is important to make sure that no trauma has occurred to the nasal cavity.



Figure 35.2: Removal of a nasal foreign body requires delicate handling with proper instruments.



CHAPTER 36

I Am Sneezing A Lot!

Dr. Abdullah Jameel Aburiziza

A 24-year-old male medical student presents with sneezing, nasal itching, clear nasal discharge, nasal congestion and headache for most of the year, but the symptoms become more severe from March to September each year for the past 6 years. He also gets a cough when the upper airway symptoms and headache are most prominent, but he says he does not have a history of asthma. Since he was 5 years old, the patient has also suffered from intermittent cough and chest wheezing, which always worsen during URTIs.

He has received courses of various oral antibiotics for clinically diagnosed sinusitis and bronchitis, and he reports that these treatments have helped the cough and headaches. The nasal symptoms have been treated as needed with oral antihistamines with moderate success. He had eczema as a child, which cleared up by the age of 7. His family history reveals that his only sibling (a 10-year-old sister) and his mother suffer from similar symptoms.

When the patient is examined in the ENT clinic, his nasal mucosa are severely congested, but no nasal polyps are noticed on endoscopic examination. The systemic examination is normal.

* * * * *

Q1. What is the most likely diagnosis?

Allergic rhinitis.

Q2. Define rhinitis.

Rhinitis is an inflammation of the mucous membranes of the nose, and it can be recognized by the symptoms of nasal stuffiness, itching, watery nasal discharge and sneezing.

or nasal stuffiness.

Q3. Classify the types of rhinitis.

Infectious:

- Viral
- Bacterial
- Other infectious agents

Allergic:

- Intermittent
- Persistent

Drug induced:

- Aspirin
- Anti-hypertensives
- Oral contraceptives
- Other drugs

Hormonal:

- Puberty
- Pregnancy
- Hypothyroidism

Other causes:

- Non-allergic rhinitis eosinophilic syndrome (NARES)
- Irritants
- Food
- Emotional
- Idiopathic
- Gastroesophageal reflux

Allergic rhinitis is an inflammatory condition of the nasal mucosa that is characterized by symptoms of pruritus, sneezing, discharge and stuffiness and is induced by an IgE-mediated response. It accounts for approximately 50% of all cases of chronic rhinitis.

Q4. What is the differential diagnosis of allergic rhinitis?

- Polyps
- Mechanical factors
 - Deviated septum
 - Hypertrophic turbinates
 - Adenoidal hypertrophy
 - Anatomic variants of the osteomeatal complex
 - Foreign bodies

– Choanal atresia

- Tumors
- Granulomas
 - Wegener's granulomatosis
 - Sarcoid
 - Infectious
- Ciliary defects
- Cerebrospinal rhinorrhea

Q5. Distinguish between intermittent and persistent rhinitis.

- Intermittent: Symptoms occur fewer than 4 days/week for less than 4 weeks.
- Persistent: Symptoms occur at least 4 days/week for more than 4 weeks.

Q6. What is the age of onset of allergic rhinitis?

The mean age of onset is 10 years old, with 80% of cases starting before age 20.

Q7. What are the major risk factors for developing allergic rhinitis?

- Family history of atopy
- Serum IgE level >100 IU before the age of 6 years
- Higher socioeconomic status
- Exposure to indoor allergens such as animals and dust mites
- Positive allergy skin test

Q8. What is the role of mast cells in the pathophysiology of allergic rhinitis?

When activated by an IgE-dependent pathway, mast cells release granule products, such as histamine, tryptase, prostaglandins and leukotrienes, into the extracellular environment. These products are associated with the development of the early-phase symptoms of nasal pruritus, sneezing, rhinorrhea and nasal blockage. The increase in these mediators is rapid and peaks within 10 to 15 minutes of allergen exposure.

In addition to the effects of acute mast cell degranulation on immediate symptom generation, mast cell degranulation contributes to the eosinophilic and basophilic mucosal inflammation that is evident in rhinitis. Mast cells in the nose contain pre-formed cytokines and chemokines that play important roles in attracting inflammatory cells, such as eosinophils, basophils and T lymphocytes to the nasal mucosa, which cause the late-phase response 4-8 hours after the initial reaction.

Q9. What are the most common triggers for seasonal and perennial rhinitis?

Seasonal: Trees, grass and mold.

Perennial: Dust mites, indoor pets (e.g., cats, dogs) and perennial molds.

Q10. List some of the main points to be reviewed when obtaining a rhinitis history.

1. Symptoms
 - What? (sneeze, itch or drip; clear

versus purulent discharge; bilateral versus unilateral congestion; associated ocular symptoms)

- When? (seasonal, age of onset)
 - Where? (indoor versus outdoor, effect of travel out of home area)
2. Triggering or exacerbating factors
 - Allergens (animals, pollen, house dust mites) (Figure 36.1)
 - Irritants (smoke, odors, fumes)
 - Miscellaneous (weather, emotions, food/alcohol)
 3. Home and occupational environmental exposures
 4. Other medical history, including medications
 5. Personal and family history of other atopic conditions (asthma, eczema, conjunctivitis)

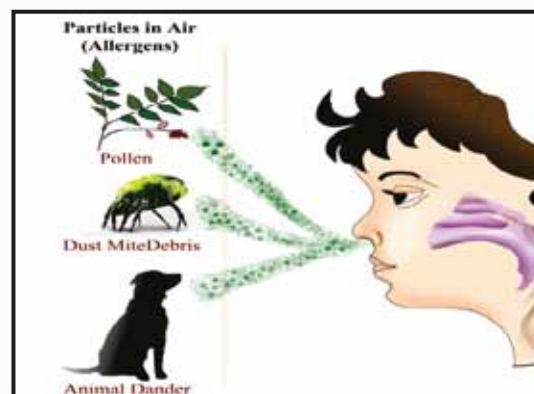


Figure 36.1: Different Allergens (pollen, house dust mites and animal)

Q11. What are the nasal polyps?

Nasal polyps are smooth, pale, semi-translucent structures composed of edematous stroma infiltrated with inflammatory cells, including activated eosinophils, activated eosinophils, lymphocytes

plasma cells and, in some cases, neutrophils. Polyps are mobile and relatively insensitive to touch. They most commonly arise from the ethmoid sinuses and are observed in the area of the middle meatus (Figure 36.2).

While nasal polyps do occur in patients with allergic rhinitis, allergy does not appear to predispose patients to polyp formation. In fact, nasal polyps are considerably more common in non-allergic patients.



Figure 36.2: Right-sided nasal cavity polyps compressing a pale-looking middle turbinate..

Q12. What is the most effective class of medication used to treat allergic rhinitis?

Intranasal steroids are significantly superior in controlling sneezing, nasal itchiness, congestion and nasal discharge compared with systemic antihistamines, montelukast and cromolyn sodium.

Q13. How do intranasal steroid sprays work?

Topical intranasal steroids have multiple effects, including the following:

- Reduction of the inflammatory cellular infiltrate in the nasal mucosa
- Reduced vascular permeability and mucous secretion
- Reduced early- and late-phase allergen responses with long duration of use

Q14. What are the potential side effects of intranasal steroid sprays?

The side effects of intranasal steroids include nasal stinging or burning, mild epistaxis and rarely septal perforation. To reduce these side effects, patients should be instructed to direct the spray slightly away from the septum.

Q15. Describe the mechanism of action of cromolyn sodium nasal spray. What is its role in the treatment of allergic rhinitis?

Cromolyn sodium inhibits the release of mediators from mast cells, which reduces the allergic reaction rather than alleviating the symptoms once they have begun. Thus, it is best to start using a cromolyn sodium before the beginning of allergy season because the onset of sustained benefit only begins after several days to 2 weeks of treatment.

Q16. Describe the mechanism of action of antihistamines. What is their role in

the treatment of allergic rhinitis?

All antihistamines competitively bind to the H₁ histamine receptor, which blocks the effects of histamine and reduces the release of mast cell mediators in response to a variety of triggers. Clinically, antihistamines reduce sneezing, itching and rhinorrhea in patients with allergic rhinitis, but they have little effect on nasal congestion.

Q17. What are the advantages of second-generation antihistamines compared with first-generation antihistamines?

Second-generation antihistamines do not cross the blood-brain barrier and do not cause sedation or reduced psychomotor performance. In addition, second-generation antihistamines have a longer duration of action and can be used once daily.

Q18. What are the side effects of oral and topical nasal decongestants?

Decongestants are alpha-adrenergic agonists that cause nasal vasoconstriction to reduce blood volume in the

venous sinusoids. The most common side effects of oral decongestants are nervousness, palpitations, loss of appetite, insomnia and urinary hesitancy. Topical products should be limited to 3-5 days of continuous use because tachyphylaxis evolves rapidly and may lead to rebound nasal congestion.

Q19. Which patients with allergic rhinitis should be considered for immunotherapy?

Patients with allergic rhinitis should be considered for immunotherapy when allergen avoidance and adjunctive pharmacotherapy have produced suboptimal results or if intolerable side effects have occurred from prolonged medication usage.

Q20. What are the possible complications of allergic rhinitis?

1. Otitis media and sinusitis
2. Decreased sense of smell
3. Sleep disturbance
4. Abnormal facial development in childhood leading to an elongated mid-face and a high-arched palate with dental malocclusion



CHAPTER 37

Nasal Congestion

Dr. Abdulaziz Al Qahtani

A 24-year-old female complains of constant bilateral nasal obstruction for 5 months that is not related to a specific time of the day. She has post-nasal drip and nasal congestion. Furthermore, her sense of smell has been impaired for almost one year. She also complains of headaches that affect her university performance and cause frequent absences.

She has no other symptoms, such as epistaxis, nasal discharge, facial pain or orbital complaints. She also has no allergic symptoms, such as itching, sneezing or rhinorrhea. She says she does not have any history of asthma or aspirin sensitivity. Her nasal examination is normal. Using a nasal speculum and headlight, there are no visible nasal lesions.

* * * * *

Q1. Is it sufficient to look with a nasal speculum to diagnose nasal disease?

No. Anterior rhinoscopy and endonasal examination are important tools for the diagnosis of nasal disease. Check for the presence of mucopurulent discharge, mucosal edema, polyps and posterior septal deviation. Evaluate the endonasal anatomy of the nasal fossa and sinuses, especially the middle meati and sphenoidal recesses.

Q2. Based on her history and examination, what is the differential diagnosis?

- a) Inflammatory lesions: chronic rhinosinusitis with or without nasal polyps, cystic fibrosis, allergic fungal rhinosinusitis and antrochoanal polyp
- b) Benign lesions: inverted papilloma, meningoencephalocele
- c) Malignant lesions: squamous cell carcinoma, adenocarcinoma, salivary gland tumor or others

Q3. Does the clinical appearance of the polyp help in making the diagnosis?

Yes, the appearance and location are important. Inflammatory polyps are usually

bilateral semi-transparent, grape-like glistening masses that originate from the ethmoid sinus (Figure 37.1). Unilateral lesions with hemorrhagic, friable or ulcerative features might alert you to the possibility of a neoplastic lesion. Masses arising from the olfactory fossa could be non-inflammatory lesions (i.e., meningoencephaloceles or olfactory esthesioneuroblastomas).



Figure 37.1: Right nasal endoscopy showing a gray pendulous semi-transparent swelling in the nasal cavity. The polyp is very different in appearance from the red middle turbinate adjacent to it.

Q4. Is there a grading system to classify the size of polyps?

Yes, the grading system helps in the evaluation of the patient and in follow-up assessments.

- a) Grade 1: small polyps within the middle meatus
- b) Grade 2: polyps within the middle meatus reaching the inferior border of the middle turbinate
- c) Grade 3: polyps extending into the nasal cavity between the inferior edges of the middle and inferior turbinates

d) Grade 4: polyps filling up the nasal cavity and extending to the floor of the nose

Q5. What is the pathophysiology of nasal polyps?

The exact cause in most cases of nasal polyposis remains uncertain. It has a multifactorial etiology resulting from dysfunction in host-environment interactions. Generally, the inflammation is mediated by T helper 2 (Th2) cells. The key cytokines are interleukin (IL)-4, IL-5 and interferon (IFN)-gamma, with a high level of tissue eosinophilia. Subsequent edema develops due to increased capillary permeability and transudation of fluid into the cellular and extracellular spaces, resulting in the formation of polyps.

Q6. List some diseases that are associated with nasal polyps.

- A. Chronic rhinosinusitis
- B. Allergic fungal rhinosinusitis (AFRS)
- C. Asthma
- D. Aspirin intolerance/salicylate sensitivity
- E. Cystic fibrosis
- F. Primary ciliary dyskinesia (including Kartagener syndrome)
- G. Churg-Strauss syndrome

Q7. Mention the most important parts of the diagnostic work-up.

- A. Endoscopic examination

- B. Radiological assessment
- C. Tissue biopsy
- D. Allergy testing
- E. Disease-specific testing, which is required for specific suspected conditions
 - Cystic fibrosis: (especially for children with nasal polyps) sweat chloride test or genetic testing
 - Aspirin intolerance: aspirin provocation test
 - Immunodeficiency screening
 - Primary ciliary dyskinesia: tissue biopsy examined under electron microscopy

Q8. What is the importance of the radiological exam?

A CT scan without contrast is the imaging method of choice to assess the severity/extent of the disease and for pre-operative evaluation of the bony surgical landmarks (Figure 37.2). Typical polyps are homogeneous and isodense, while fungal polyps have heterogeneous hyperdense foci (double-density signatures secondary to heavy metal production by the fungi). An MRI is indicated in cases of bone erosion, skull base destruction or orbital in-



Figure 37.2: Coronal CT scan of the paranasal sinuses showing homogeneous soft tissue density involving the maxillary and ethmoid sinuses. (Picture courtesy of Professor Paolo Castelnovo)

Q9. When do we need to perform a tissue biopsy?

1. Atypical presenting symptoms
2. Abnormal endoscopic findings and/or radiological appearance
3. Unilateral nasal mass

A CT scan with contrast and/or an MRI with gadolinium are important before polyp biopsy to rule out a vascular lesion and meningoencephalocele, which are contraindications for biopsy.

Q10. Allergy testing is important for which cases?

It is important when the patient has a history of asthma or allergic rhinitis or has allergic fungal sinusitis. A skin test is the preferred method.

Q11. What is the management strategy for nasal polyps?

There is no definitive treatment protocol for nasal polyps; therefore, multimodal therapy is needed and should be tailored specifically to each patient's condition. A course of medical treatment must be prescribed before any surgical decision is made. Identification of the predisposing factors and the possible etiology are important in the management.

Q12. List the main medical modalities of treatment.

1. Intranasal steroids: These agents are associated with reduced or delayed growth of small nasal polyps and the prevention of post-operative recurrence. Safety for both short- and long-term use has

been observed.

2. Oral steroids: Oral steroids are indicated in the following conditions:

- As part of maximal medical treatment of massive polyps before considering surgery
- During the perioperative period to reduce inflammation and augment healing post-operatively
- As part of the management of comorbid conditions such as asthma and allergic rhinitis

They must be used with caution in patients with gastrointestinal ulcers, diabetes mellitus (DM), cataracts, glaucoma and osteoporosis.

3. Leukotriene modifiers: These agents prevent or reverse some of the pathological features associated with the inflammatory processes mediated by leukotrienes.

4. Other agents used in certain conditions include the following: saline wash, macrolide antibiotics, antihistamines, antifungals and aspirin desensitization therapy.

Q13. When is surgery indicated?

Functional endoscopic sinus surgery (FESS) is indicated for nasal polyps

that do not respond to medical treatment or are associated with complications.

Q14. If nasal polyps are left untreated, what are the complications?

A. Intranasal

- Recurrent sinusitis
- Chronic sinusitis
- Acquired nasal deformity

B. Orbital

- Proptosis
- Diplopia
- Blindness

C. Intracranial

- Meningitis
- Encephalitis

D. Respiratory

Obstructive sleep apnea

Asthma flare-ups

Q15. Is this disease treatable? What is the prognosis?

Outstanding short- and long-term results have been observed after surgery. Symptoms improve in approximately 90% of patients following FESS, which significantly improves quality of life. Recurrence can occur in some cases that are difficult to treat, and these cases require special testing and management.

CHAPTER 38

Nose Deformity

Prof. Sameer Ali Bafaqeeh

An 18-year-old male complains of a nose deformity. He was involved in a road traffic accident 10 years ago. After the accident, he started to have left nasal obstruction and developed an external nasal deformity and a skin scar below the nose. He was told that surgery on the nose should not be performed before the age of 18. He is very embarrassed by the shape of his nose, which interferes with his activities and education. He has decided that he will not go to university before fixing this major problem.

On examination, he has a clear C-shaped nasal deformity with a deviation of the nasal septum toward the left side. He has dry mouth and a prominent bat-like left ear but an otherwise normal ENT examination (Figures 38.1A and 1B).

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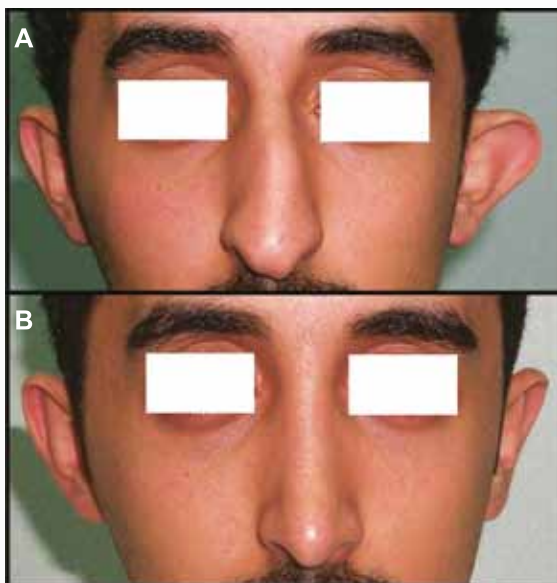


Figure 38.1: A. Pre-op B. post-op examination.

Q1. Why is there an increase in the number of patients looking for cosmetic surgery?

Good outcomes, safe surgeries and greater awareness and concern about facial appearance have caused an increase in the number of patients who seek corrective esthetic facial surgeries and spend a great deal of time and money on cosmetic procedures. This trend can be observed in the beauty centers that provide services ranging from holistic medicine to facial- and

procedures. Otolaryngologists are expert facial surgeons who also treat related functional problems, particularly problems related to the nose.

Some patients with obvious deformities are embarrassed about requesting corrective operations, while other patients exaggerate a minor cosmetic deformity almost to the point of obsession. It is vital to evaluate the patient's psychological and physical profiles to ensure that they have realistic expectations for what can be accomplished. This understanding is essential to avoid dissatisfaction, and the opinions of a psychologist and psychiatrist may be useful.

Q2. What are the options for treating his scar?

Skin lesions are common in the face and may be situated in awkward places (e.g., the canthus of the eye or the nasal tip). Accurate pre-operative diagnosis of the lesion is imperative in determining the optimal treatment. There are many modalities to remove cutaneous lesions besides excision: freezing, cautery/electrosurgery, curettage, lasers and ionizing radiation.

Q3. When is biopsy indicated?

Where there is any doubt regarding the histology, a biopsy either from the edge of the lesion or via a complete excision is preferable to destructive removal. Excision of lesions should be performed with the incisions placed in the

relaxed skin tension lines of the face (Figure 38.2). This approach produces the least tension on the repair and the best cosmetic result. Larger defects that cannot be closed by undermining and advancement of the edges require a flap or free skin graft (Figure 38.3).

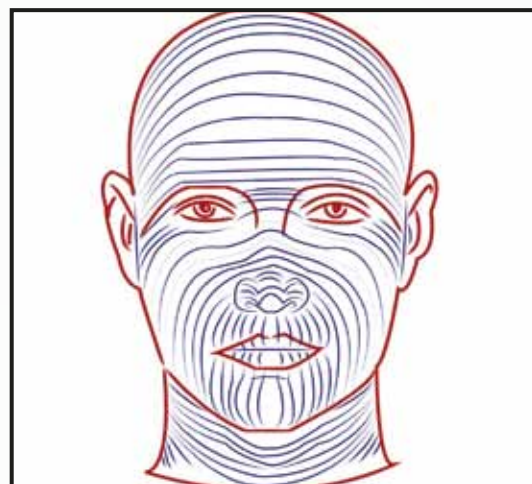


Figure 38.2: Relaxed skin tension lines for the excision of facial lesions.

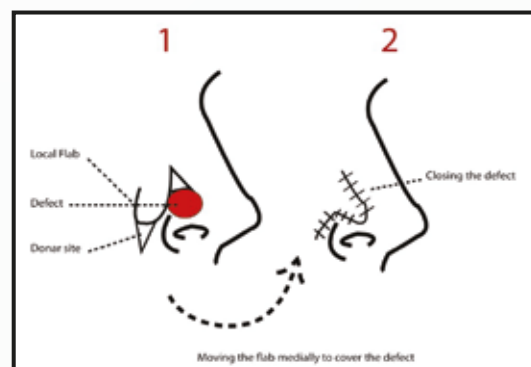


Figure 38.3: A flap can be used to close a skin defect in the face.

Q4. The patient wants laser surgery. Do lasers have any role in facial cosmetics?

Laser surgery has gained popularity recently for facial lesions and for resurfacing areas of the face. The different types of lasers (based on wavelength)

The CO₂ lasers vaporize skin, and when combined with an oscillating beam, they can precisely remove tissue to a calculated depth, allowing regeneration from the adnexa. Pigmented lesions (e.g., telangiectasias) absorb the green light of an argon laser. A KTP or pulsed-diode laser can produce selective thrombosis in vessels.

Q5. Is it true that rhinoplasty surgery is only performed for women? How easy is this surgery?

Rhinoplasty is one of the most challenging operations in facial plastic surgery. Its main purpose is to maintain a balance between form and function. There are many variations in nasal form that can be altered by trauma or disease. Patients request rhinoplasty for a variety of reasons: Women often want it because their nose is too large, usually with a dorsal hump (Figures 38.4A and B), and men frequently request it because of a combination of functional and cosmetic problems, which are often trauma related (Figures 38.5A, B, C, D, E and F).

A deviated nose is typically associated with a septal deformity, and it is essential to identify and correct the septal de-

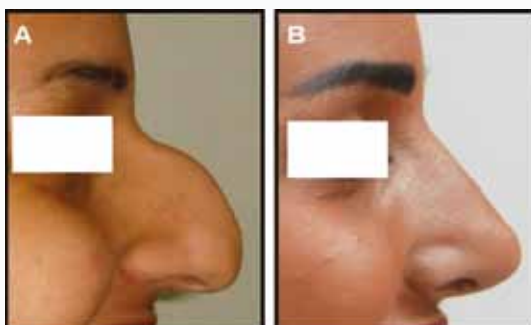


Figure 38.4: A: Pre-operative and B: post-operative examination.

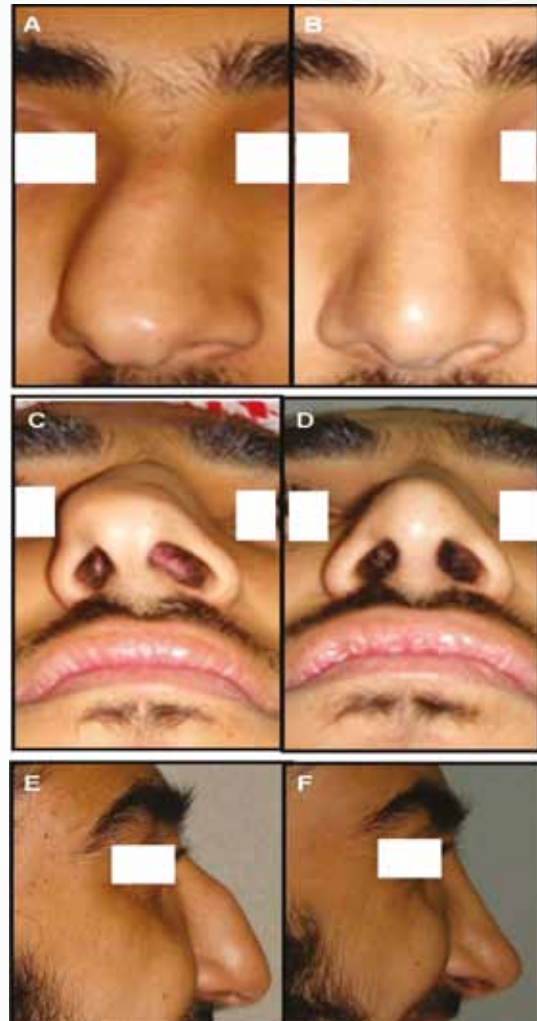


Figure 38.5: A,B,C: Pre-op nose pictures; D,E,F: post-op nose pictures

Q6. What are the purposes of rhinoplasty?

Rhinoplasty has two fundamental purposes. It should restore a normal shape to the nose, so it harmonizes with the rest of the face. In addition, the function of the nose and nasal sinuses with regard to respiration, olfaction, etc., should be maintained, improved and returned to normal.

Q7. Part of the nose is bony, and part of it is cartilaginous. Is there a difference between how they are treated during rhinoplasty?

Anomalies of the cartilaginous part of the nose include the following: a nasal tip that is hanging (Figure 38.6), too flat or too wide (bulbous, boxy) (Figures 38.7A and B), cleft (Figures 38.8A and B) or too short; nasal alae that are flaccid, too arched or asymmetrical; or a nasal columella that projects too much (hanging) (Figure 38.9) or is too retracted (Figure 38.10), thick, short or bent. The entire nose may also be too long or too short. Anomalies of the bony part of the nose include the following: a hump (Figure 38.11), a saddle nose (Figure 38.12), a twisted nose (Figure 38.13), a too broad nose (Figures 38.14 A and B) or too narrow nose.

The bony and cartilaginous parts of the nose usually need to be corrected together.

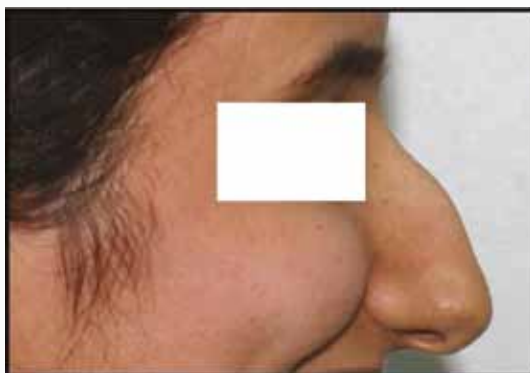


Figure 38.6: Hanging collumela & droopy nose.



Figure 38.7A & B: Pre & Post-op result of boxy nasal tip.

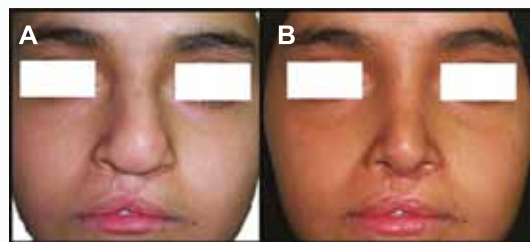


Figure 38.8A & B: Pre- & post-op results for cleft lip secondary to nasal deformity.

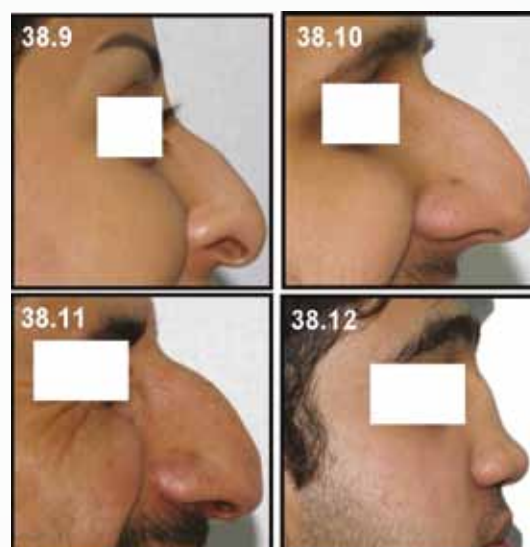


Figure 38.9, 38.10, 38.11 & 38.12 : Cases.



Figure 38.13, 38.14A & B: Cases.

Q8. What are the common surgical approaches in rhinoplasty?

Three approaches have proven successful for rhinoplasty:

1. The splitting approach
2. The delivery approach
3. The open approach (Figure 38.15)

The selected approach depends on the surgeon's skill and the specific morphological problems. Thick skin predisposes the patient to healing problems, while thin skin limits the selection of surgical techniques (e.g., using tip or shield grafts that might be visible through the skin).

Most refinement and hump-reduction rhinoplasties are performed as closed procedures (i.e., the incisions are entirely within the nasal fossa).

For a crooked nose, traumatic injury, droopy asymmetric nasal tip, and nasal deformity secondary to cleft lip and palate, most of the revision cases and marked caudal and dorsal deviated nasal septum are better to be corrected via an open septorhinoplasty approach which has an external incision across the columella allowing an extended range of surgical options (Figure 38.16 shows some of these surgical techniques).

Weak or under-projecting chins should be augmented via an implant to improve the profile's appearance. This approach often produces better balance and allows a more conservative reduction of the nose. (Figures 38.17A, B, C and D).



Figure 38.15: Open approach for rhinoplasty



Figure 38.16: Surgical techniques



Figure 38.17: Open approach for rhinoplasty

Q9. The patient is also interested in fixing his left ear deformity.

Prominent ears are a source of ridicule for many children, and patients often feel self-conscious in adult life. The deformity is usually a combination of a deep concha bowl and failure of the anti-helix to develop. The latter, if recognized at birth, can be treated with taping over a splint within the first 5 days. Otherwise, correction is best left until just before school entry but can be considered at any age after approximately 3.5 years. Various techniques using cartilage scoring and suturing are employed, usually under general anesthesia

for children, and the bandages are removed after approximately 10 days. A sweatband is worn at night for another 2-3 weeks. Otoplasty in an adult, particularly unilateral otoplasty, can be performed under local anesthesia, which would be the case for our patient (Figures 38.18A and B).

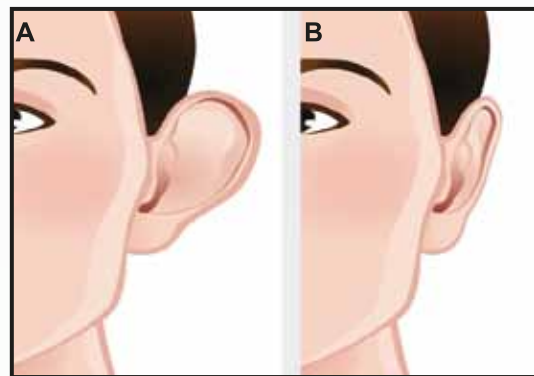


Figure 38.18A & B: Otoplasty in an adult.

CHAPTER 39

Ear Fullness

Prof. Saeed Al Ghamdi

A 3-year-old child with a known case of congenital cleft palate presents with a history of recurrent ear discharge and hearing loss for more than 1 year. A detailed history reveals left ear mucopurulent discharge every 3-4 weeks with no fever and no association with URTIs. The child's history is significant for a previous palatal repair surgery when he was 2 years old. The family history is irrelevant. A systemic review reveals no other associated anomalies.

During the clinical examination, the child is afebrile, and his general condition is good. An oral examination reveals a previous surgical scar and normal palate. An ear examination shows a left ear drum perforation and right middle ear effusion.

* * * * *

Q1. Describe the anatomy of the Eustachian tube.

The tube is divided into a pharyngeal portion (cartilaginous) and a tympanic portion (bony) and is lined with ciliated columnar epithelium in the cartilaginous portion and cuboidal epithelium in the bony portion.

The average length is approximately 36 mm, and the cartilaginous portion is approximately 24 mm. The junction of the two portions is the narrowest point and is called the isthmus. In children, the tube is more horizontal and shorter compared with adults.

Q2. What are the functions of the Eustachian tube

1. Regulation of middle ear pressure

2. Drainage of middle ear secretions
3. Protection of the middle ear from nasopharyngeal secretions (Figure 39.1)



Figure 39.1: Eustachian tube drains the middle ear secretions to the nasopharynx.

Q3. List the possible intrinsic factors that may cause Eustachian tube dysfunction.

1. Congenital causes such as cleft palate, which can cause a muscular defect

2. Abnormal patency (patulous Eustachian tube)
3. Functional immaturity
4. Acute and chronic salpingitis

Q4. What are the possible extrinsic factors that may cause Eustachian tube dysfunction?

1. Enlarged adenoids
2. Palatal dysfunction
3. URTIs
4. Nasopharyngeal neoplasm

Q5. Write a short note on a patulous Eustachian tube.

This condition results from a patent tube secondary to rapid weight loss, pregnancy, pharyngitis or a chronic wasting disease. Patients commonly present with a roaring sensation in the ear that is synchronous with respiration, autophony and fullness of the ears.

Examination reveals tympanic membrane movement in and out during respiration. It is easily illustrated via the Valsalva maneuver.

Treatment should target the underlying disease. A tympanostomy tube may help treat the symptoms.

Q6. Name the muscles associated with the Eustachian tube and their nerve supply.

- Levator veli palatini (vagus nerve)
- Salpingopharyngeus (vagus nerve)
- Tensor tympani (mandibular nerve branch of the trigeminal nerve)
- Tensor veli palatini (mandibular nerve branch of the trigeminal nerve)

Q7. How do you evaluate Eustachian tube dysfunction?

1. Valsalva maneuver
2. Tympanometry (a blocked tube is usually associated with a type B or C tympanogram)
3. Flexible endoscopic examination
4. Radiological evaluation (lateral neck X-ray)

Q8. What is the importance of unilateral Eustachian tube dysfunction?

Nasopharyngeal tumors should be excluded.

Q9. What is acute salpingitis (acute tubal catarrh)?

Infection of the epithelium of the tube can cause congestion, swelling and excessive secretions, which leads to tube obstruction and the development of acute suppurative otitis media. Patients may present with blocked ears and pain, and the condition is commonly observed in patients with the common cold. Most cases resolve spontaneously. Decongestants may be useful.

Q10. What is chronic salpingitis?

Chronic salpingitis usually results from chronic tube obstruction as in chronic non-suppurative ear disease or adhesive otitis media. Patients mainly present with blocked ears and hearing loss, and ear examination may reveal a retracted drum with possible effusion.

The treatment, in addition to ventilating the tubes, is to ensure that nasopharyngeal pathology is ruled out.