

Diagnosis in Otorhinolaryngology

An Illustrated Guide

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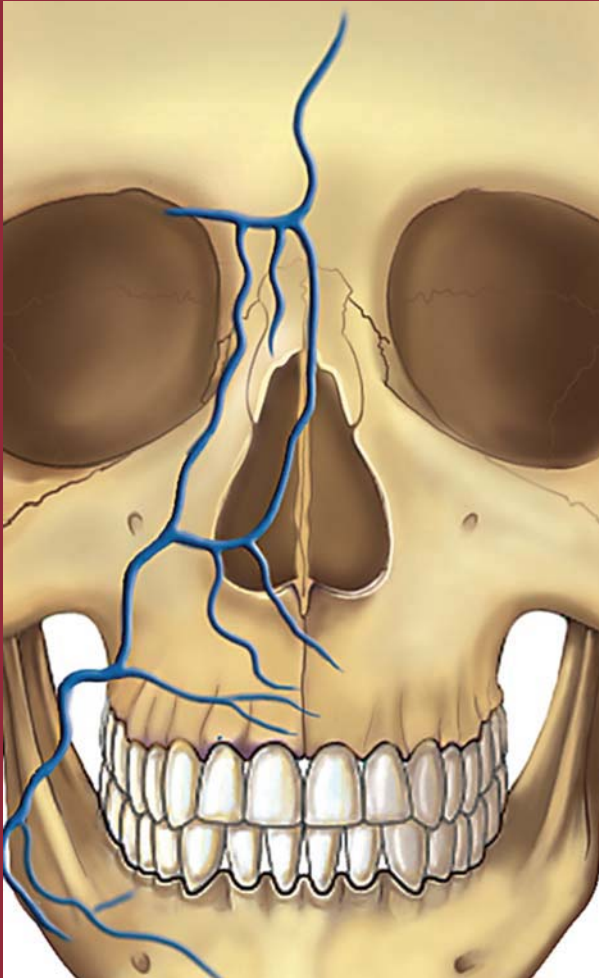
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2.1

The Common Cold and the Flu

Both the flu and the common cold are respiratory illnesses caused by different viruses. The flu is an infection of the respiratory system caused by the influenza virus, the common cold is caused mainly by rhinoviruses. Common cold/acute viral rhinosinusitis is defined as duration of symptoms for less than 10 days. Acute nonviral rhinosinusitis is defined as an increase of symptoms after 5 days or persistent symptoms after 10 days with less than 12 weeks' duration.

Although the symptoms are similar and it is difficult to tell the difference between the common cold and the flu based on symptoms alone, the flu is worse than the common cold. There is usually fever (temperature above 39°C), and symptoms such as headache, body aches, extreme tiredness, sore throat, and dry cough are more common and intense. The symptoms appear suddenly. People with colds are more likely to have a runny or stuffy nose. Colds generally do not result in serious health problems, such as pneumonia, bacterial infections, or hospitalizations. Adults get an average of two to four colds per year, mostly between September and May. Young children suffer from an average of six to eight colds per year. People recover from the common cold in 1 week; however, recovery from the flu may take more than 1 week, especially for the elderly, who may feel weak for a long time even after symptoms resolve.

Nonsteroidal anti-inflammatory drugs (NSAIDs) can be used. However, acetaminophen, aspirin, or any other NSAIDs may worsen asthma and/or peptic ulcers. Aspirin should not be used in children younger than 18 years because it may play a role in causing Reye syndrome, a rare but severe liver and central nervous system condition. Congestion, cough, and nasal discharge may be treated with a decongestant, antihistamine, or a combination of the two. Some people such as those with thyroid disease or high blood pressure should not take decongestants. There are no antiviral medications available for treating the common cold. Antibiotics are not useful for treating a cold, and should only be taken to treat bacterial complications that arise from it.

Other Remedies

Herbs and minerals such as echinacea, eucalyptus, garlic, honey, lemon, menthol, zinc, and vitamin C have received extensive publicity as cold remedies. However, none of these claims is solidly supported by scientific studies.

Adequate liquid intake (eight glasses of water and/or juice per day) is recommended to prevent dehydration. Coffee, tea, or cola drinks that contain caffeine and any drinks that contain alcohol should be avoided to prevent their dehydration effects. Smoking should be stopped. Since inhaling smoke of other smokers will cause more irritation in the throat and will increase coughing, patients should stay away from other smokers.

Bed rest is helpful for recovery. Until the symptoms are gone, it is not advisable to go back to full activity. In the treatment of the flu, antiviral medications may be used. They may reduce the duration of the disease if started early. Oseltamivir or zanamivir may be used to treat the influenza virus. Oseltamivir or zanamivir given within 2 days of the appearance of flu symptoms will reduce the length of the illness and the severity of symptoms by at least 1 day. Early treatment can lead to faster recovery.

Flu Vaccines May Help to Prevent Getting the Flu

There are currently two vaccine options: the flu shot and the nasal spray vaccine. The shot gives more reliable protection and the spray is recommended only for non-high-risk groups.

The best tool for preventing the flu is the flu vaccine, and the best time to get a flu vaccine is from early October to mid-November. The vaccine can also be given at any point during the flu season, even if the virus has already begun to spread in the community. A flu vaccine should be repeated every year because the virus is constantly changing and new vaccines are developed annually to protect against new strains.

Table 2.1.1 Who should get a flu vaccine?

Adults 50 years or older
All children under 5 years of age (only after 6 months of age)
Adults and children aged 2–64 years with chronic medical conditions, especially asthma, other lung diseases, and heart disease
All women who will be pregnant during the influenza season
Residents of nursing homes and other chronic care facilities
Health-care workers involved in direct patient care

Table 2.1.2 Contraindications to flu vaccination

Egg allergy
History of Guillain-Barre syndrome
Acute illness or fever

Table 2.1.3 Clinical features of the common cold and flu

	Common cold	Flu
Virus	Rhinovirus	Influenza
Contagiousness	Droplets by inhalation or touch	Droplets by inhalation
Onset	1–3 days after virus entrance	Sudden
Duration	One week	One week or more
Frequency	Children six to eight colds per year, adults two to four colds per year	Once
Symptoms	Milder Weakened senses of taste and smell, cough, runny or stuffy nose, sneezing, scratchy throat	Worse Fever (39°C or above), body aches, extreme tiredness, dry cough more common, headache, sore throat, chills, tiredness
Complications	No serious complications	May have serious complications, pneumonia, bacterial infections May be fatal in elderly, immunocompromised, and chronically ill patients
Treatment	Acetaminophen Antihistamine and/or decongestant Adequate fluid intake (eight glasses of water or juice) Avoid smoking and alcohol Avoid caffeine and alcohol No antibiotics	Acetaminophen Antihistamine and/or decongestant Adequate fluid intake (eight glasses of water or juice) Avoid smoking and alcohol Avoid caffeine and alcohol No antibiotics

Table 2.1.4 How to prevent a cold

Close contact with people who have a cold should be avoided especially during the first few days when they are most likely to spread the infection
Hands should be washed after touching someone who has a cold
Fingers should be kept away from the nose and the eyes to avoid self-infecting the cold virus particles
A second hand towel should be put in the bathroom for healthy people to use
The environment should be humidified
The nose and the mouth should be covered with a tissue when coughing or sneezing

2.2

Rhinitis

Rhinitis is a clinical diagnosis and is defined as inflammation of the nasal mucosa with one or more symptoms of sneezing, itching, rhinorrhea, and nasal blockage lasting for at least 1 h on most days. All diseases causing rhinorrhea and nasal obstruction should be considered in the differential diagnosis of rhinitis.



Fig. 2.2.1 Allergic rhinitis. Serous nasal discharge with hypertrophic, pale inferior turbinates

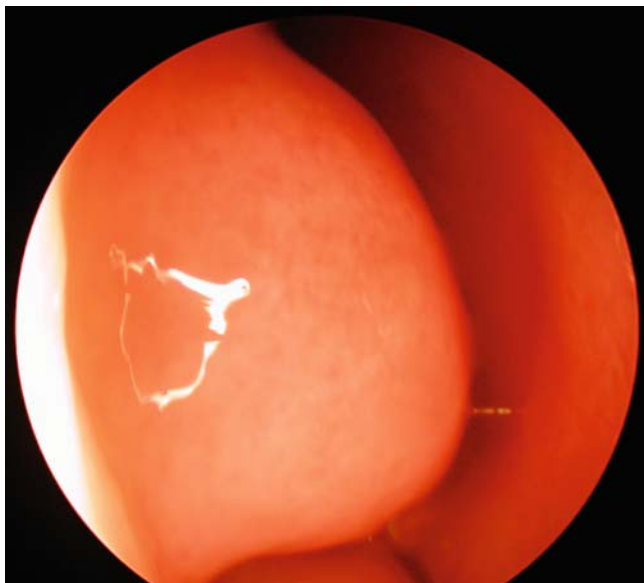


Fig. 2.2.2 Acute rhinitis, early period. Right inferior turbinate mucosa is hyperemic and there is serous secretion

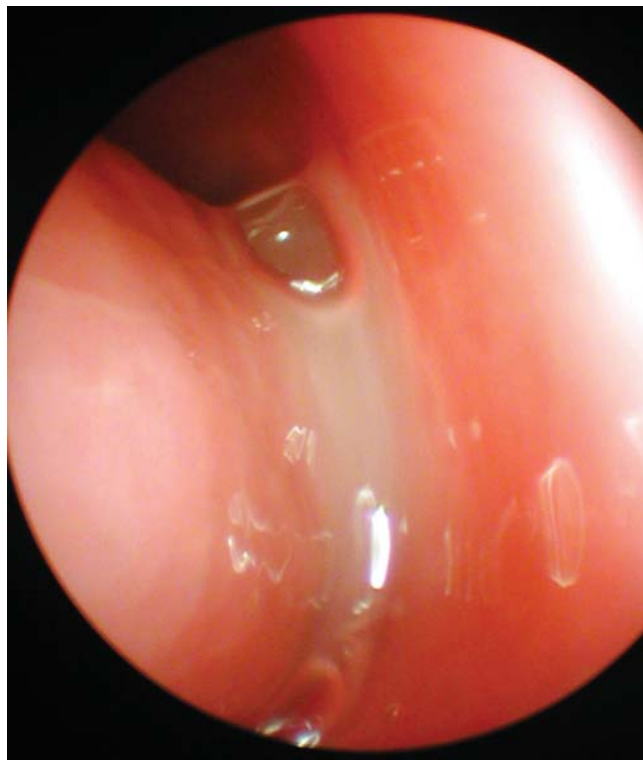


Fig. 2.2.3 Upper respiratory tract infection, 6th day, mucoid nasal discharge

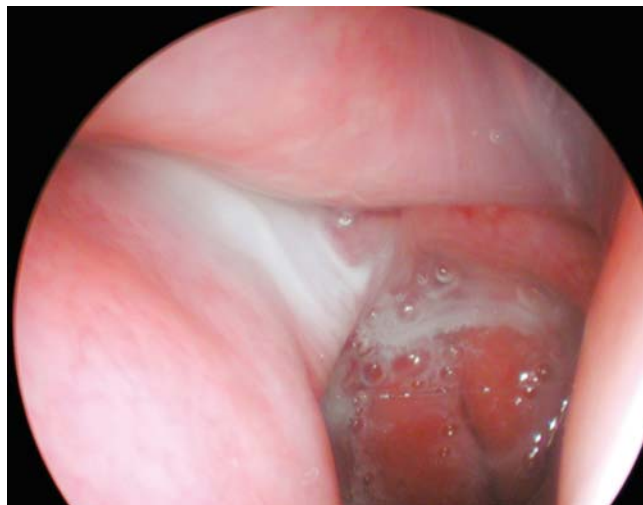


Fig. 2.2.4 Right acute maxillary sinusitis. Purulent nasal discharge with draining to the nasopharynx through the middle meatus



Fig. 2.2.5 Chronic sinusitis, purulent discharge in left nasal cavity



Fig. 2.2.6 Eosinophilic mucin in a patient with nonallergic rhinitis with eosinophilia syndrome (NARES). The discharge is sticky, thick, and yellow-green

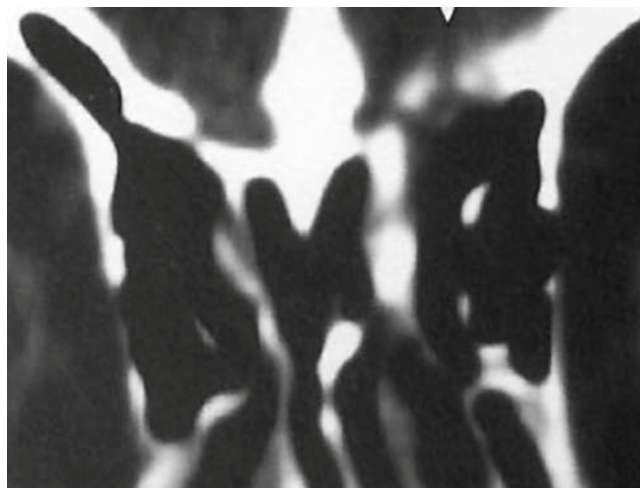


Fig. 2.2.7 Cerebrospinal fluid (CSF) rhinorrhea. Coronal CT showing fracture line in the fovea ethmoidalis of anterior ethmoid area

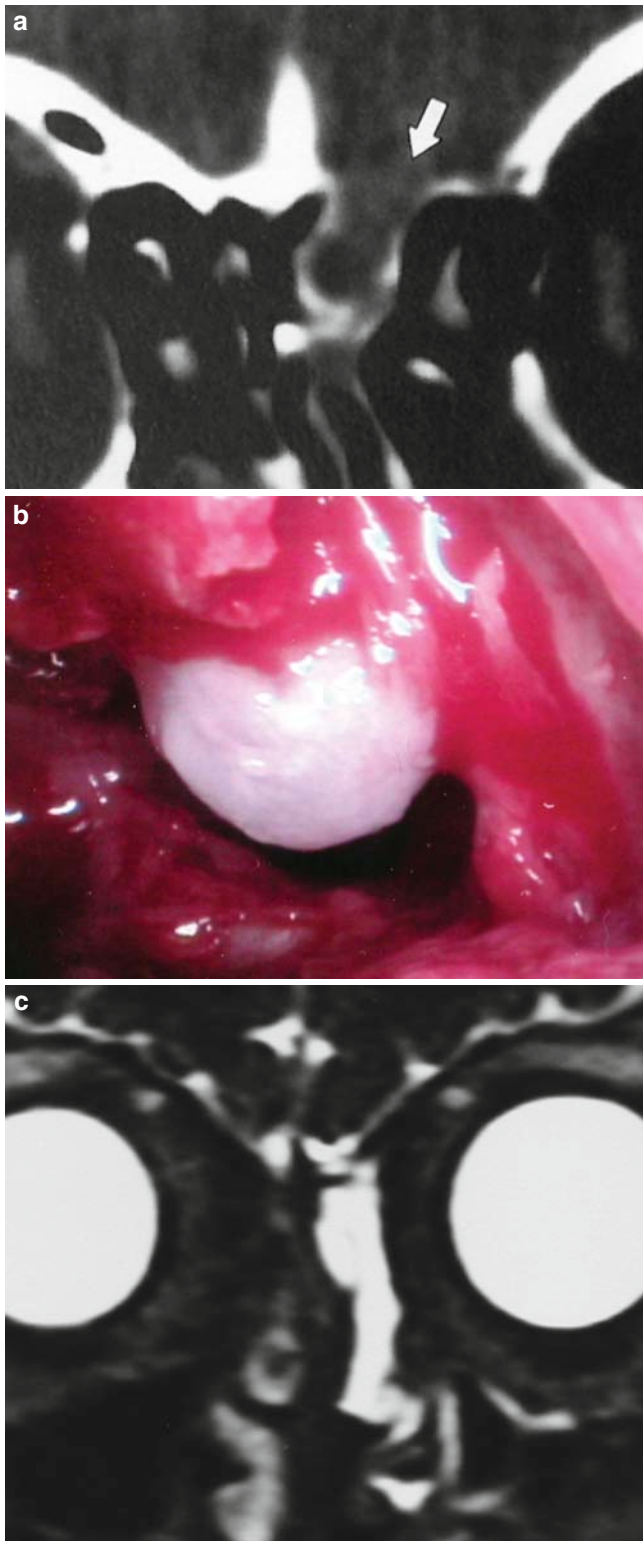


Fig. 2.2.8 CSF rhinorrhea. (a) Coronal CT showing herniation through lamina cribrosa at the anterior ethmoid area (arrow). (b) Photograph of the herniated tissue. (c) MR cisternography; CSF leak is seen

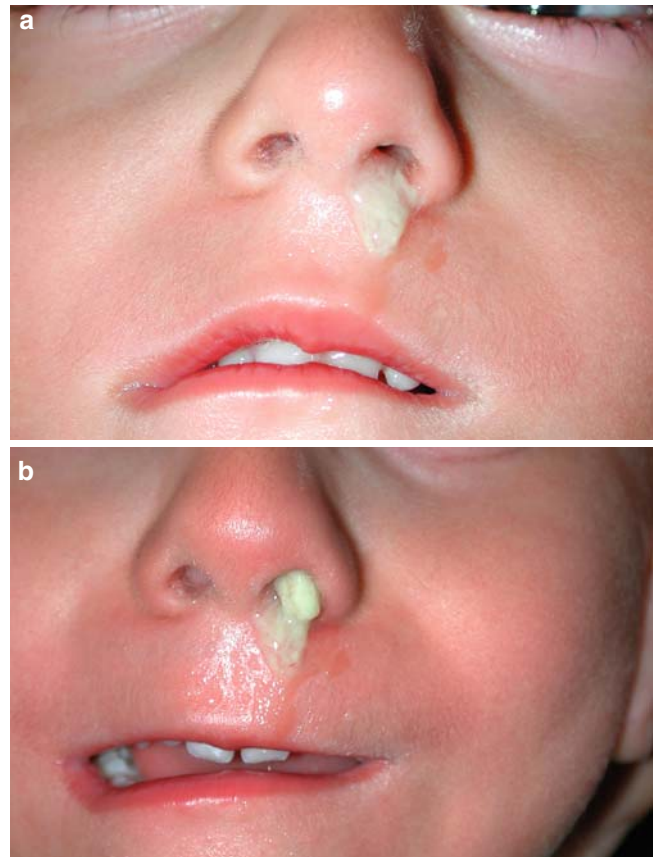


Fig. 2.2.9 Nasal foreign body. (a) Unilateral left-sided purulent nasal discharge; the patient presented with a 1-month history of foul odor. (b) Foreign body, a piece of tissue, is seen in the left nostril during removal. Generally young children insert foreign bodies into their noses. A unilateral nasal discharge in children should raise the suspicion of a foreign body. Foul smell can be noticed in patients if the foreign body is present for a long period of time. To remove the foreign body, the child should be immobilized. The limbs may be wrapped in a linen cloth and the head kept immobile. Ithar sonde may be used to remove the foreign body. Its blunt curved end passes behind the foreign body and is then taken out. General anesthesia may be given in certain situations, since attempts at removal may push the object to the nasopharynx with the risk of inhalation



Fig. 2.2.10 A rubber foreign material in the left nasal passage. The child should be kept immobile during removal of the foreign body. To remove the foreign material, a curved instrument with a blunt tip should be used such as Ithar sonde or ear curette



Fig. 2.2.11 Atrophic rhinitis. Atrophy of the nasal mucosa and turbinates with yellow-green copious foul-smelling crusts filling the nasal cavity

Table 2.2.1 Classification of rhinitis^a

<i>Allergic</i>
Seasonal
Perennial
<i>Infectious</i>
Acute
Chronic
Specific
Nonspecific
<i>Nonallergic</i>
Eosinophilic rhinitis (NARES – nonallergic rhinitis with eosinophilia syndrome)
<i>Others</i>
Occupational
Hormonal
Drug induced
Irritants
Emotional
Food (gustatory rhinitis)
Atrophic
Geriatric
Idiopathic

^aInternational Consensus Report on the Diagnosis and Management of Rhinitis 1994

Nonallergic rhinitis with eosinophilia syndrome (NARES) is a type of rhinitis associated with the symptoms of perennial rhinitis without any identifiable allergen hypersensitivity. IgE-mediated mechanisms do not play a role. Excessive eosinophilia is demonstrated in nasal secretions. Nonallergic asthma and analgesic intolerance are more common in these patients. The etiology is unclear. They often respond well to treatment with intranasal corticosteroids.

Occupational rhinitis arises in response to an airborne agent present in the workplace. Causes include laboratory animals, hair (hairdressers), grain (bakers and agricultural workers), wood dusts, latex, and chemicals.

Hormonal rhinitis can occur during pregnancy, puberty, and also in hypothyroidism and acromegaly. Postmenopausal hormonal changes may also cause atrophic nasal pathologies.

Emotional rhinitis is the result of emotional factors such as stress and sexual arousal due to autonomic stimulation as in honeymoon rhinitis.

Gustatory rhinorrhea occurs when eating hot and spicy foods. True food allergy never produces isolated rhinitis symptoms. Hypersensitivity reactions to colorants and preservatives in the food may also occur.

The term idiopathic rhinitis is generally used instead of vasomotor rhinitis. Vasomotor rhinitis is a subgroup of NARES, which is thought to be due to an imbalance of autonomic nervous supply and peptidergic nervous mechanisms. Engorged blood vessels lead to nasal obstruction. These patients present with nasal hyperresponsiveness to non-specific stimuli such as strong smells, irritants such as exhaust fumes, or environmental temperature. Nonimmunologic stimuli such as cold air can degranulate mast cells with mediator release and may cause the symptoms.

Atrophic rhinitis is characterized by progressive atrophy of the underlying bone of the turbinates and nasal mucosa. Copious foul-smelling crusts fill the nasal cavity. The patient complains of hyposmia, nasal congestion, and constant bad smell in the nose. *Klebsiella ozaenae* is generally found in the nasal cavity of these patients.

Table 2.2.2 Differential diagnosis of rhinitis

Polyps
<i>Mechanical factors</i>
Septum deviation
Turbinate hypertrophy
Adenoidal hypertrophy
Foreign bodies
Choanal atresia
Tumors
Benign
Malignant
Granulomas
Wegener granulomatosis
Sarcoidosis
<i>Infectious</i>
Tuberculosis
Lepra
Malignant midline destructive granuloma
CSF fistula

Table 2.2.3 Diagnostic tests for rhinitis

Skin prick tests
Specific IgE measurements
Nasal smear
Nasal provocation tests
Histamine/methacholine
Allergen
Rhinomanometry
Acoustic rhinometry
CT, MR imaging
Biopsy, electron microscopic examination
Sweat test

Table 2.2.4 Drugs that can induce rhinitis

Antihypertensives
Reserpine
Guanethidine
Phentolamine
Methyldopa
ACE inhibitors
Alpha adrenoreceptor antagonists
Topical ophthalmic beta blockers
Chlorpromazine
Aspirin
Nonsteroidal anti-inflammatory agents
Oral contraceptives
Topical decongestants (rhinitis medicamentosa – long-term use of cocaine and nasal drops or sprays)

Table 2.2.5 Diagnostic features of noninfectious rhinitis

	Seasonal	Perennial	Perennial nonallergic
Time of year	Seasonal	Perennial	Perennial
Age of onset	10–20	10–20	Adulthood
Prominent symptom	Rhinorrhea, sneezing, itching	Rhinorrhea, sneezing, itching	Rhinorrhea, blockage
Eye symptoms	Common	Uncommon	Not present
Nasal cytology	EO (Eosinophil)	EO	EO/NT (Neutrophil)
Allergens	Pollens	Dust mite, moulds, animal	Negative
Polyps	Uncommon	Uncommon	Frequent

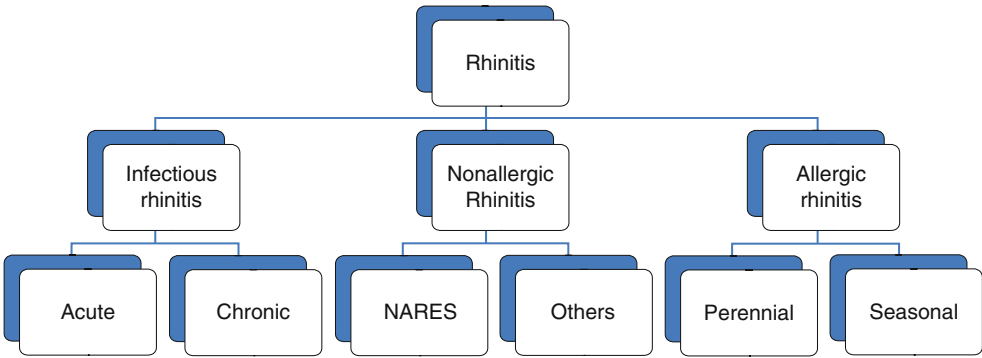


Fig. 2.2.12 Classification of rhinitis

2.3

Allergic Rhinitis

Allergy is an inappropriate and harmful immune response to a normally harmless substance. Generally, allergens are proteins that do not cause any reactions in nonatopic individuals.

What is atopy?

Atopy is an inherited predisposition to produce IgE antibodies to certain substances.

What is the difference between atopy and allergy?

Atopy is the genetic predisposition to produce IgE antibodies. To develop allergy, stimulation of the cells of the immune system to produce IgE antibodies is needed. Although almost 25–30% of the population is atopic, not every atopic person develops allergy. Environmental factors are important in the development of allergic disease.

Allergic Reaction

Allergens enter the body through the airways, the gastrointestinal tract, or the skin. In atopic patients an allergen is recognized as being foreign to the immune system. B cells are stimulated to produce specific IgE antibodies. These IgE antibodies bind to the surface of mast cells. On subsequent exposures, the allergens bind to the IgE antibodies. Bridging two IgE antibodies makes the mast cell degranulate and the mast cell releases histamine and other cytokines that cause allergic reactions.

Early Response

The early response is initiated after bridging of IgE antibodies on the mast cells. Mast cells release mediators such as histamine, prostaglandins, leukotrienes, platelet-activating factor, and bradykinin. These mediators cause vascular dilatation, increased permeability, and attract inflammatory cells into the tissues starting the inflammation. The early response is characterized by sneezing, rhinorrhea, bronchoconstriction, and increased bronchial responsiveness.

Late Response

The mediators released from mast cells attract inflammatory cells such as eosinophils, lymphocytes, neutrophils, and monocytes into the tissues. Therefore, the late response is a cell-mediated response. The late response is characterized by prolonged mucus secretion, edema formation, and bronchial hyperresponsiveness.

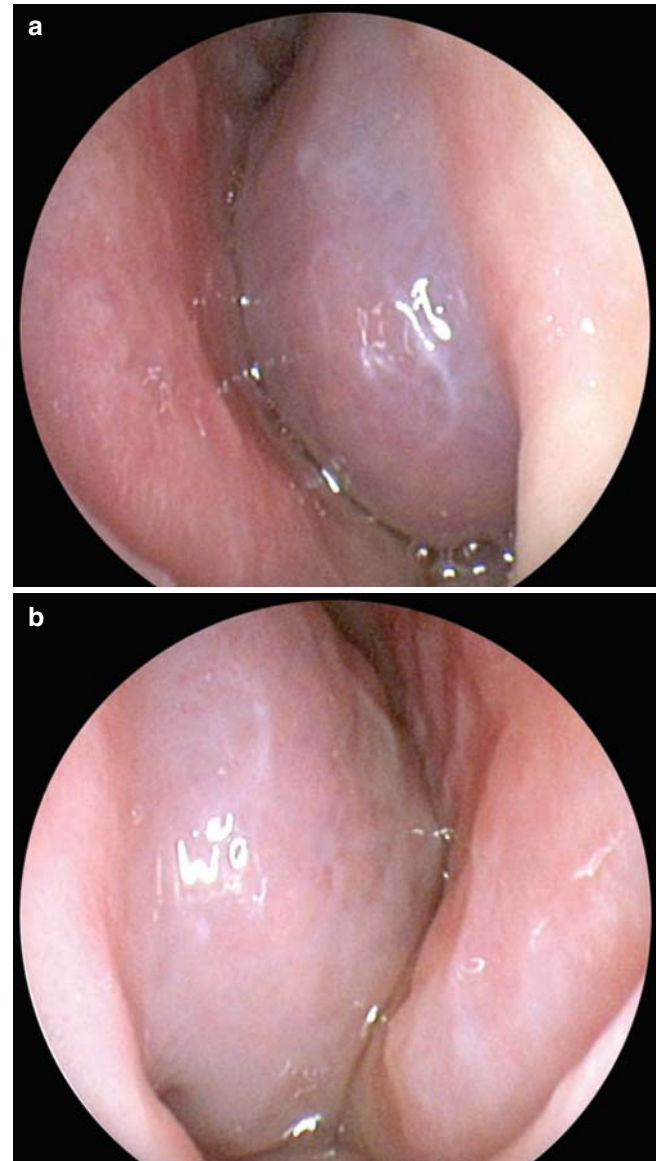


Fig. 2.3.1 (a, b) Allergic rhinitis. The turbinates are *pale, bluish*, and swollen. Watery serous secretion is seen in both nasal passages



Fig. 2.3.2 Allergy salute is a very common sign of allergic rhinitis in children



Fig. 2.3.5 Skin prick test



Fig. 2.3.3 Supratip crease. Horizontal line in the supratip area due to repeated use of allergic salute

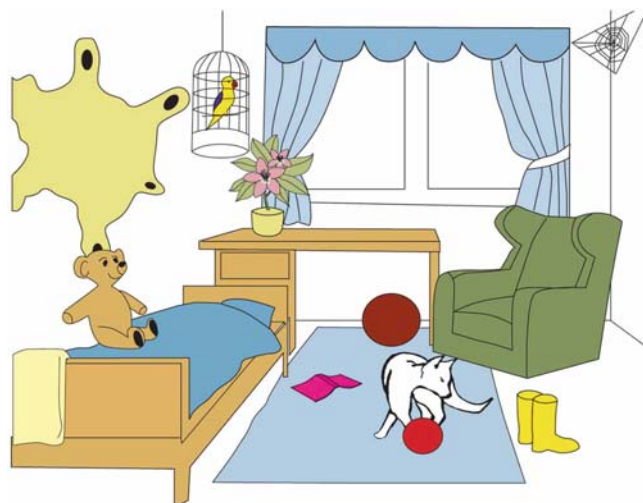


Fig. 2.3.6 Items that should not be present in the room of an allergic child



Fig. 2.3.4 Long, silky eyelashes in an allergic child



Fig. 2.3.7 Allergic conjunctivitis, erythema, and edema of the conjunctival mucosa, and watering. Limbal elevation can be identified (courtesy of Kiratlı D)

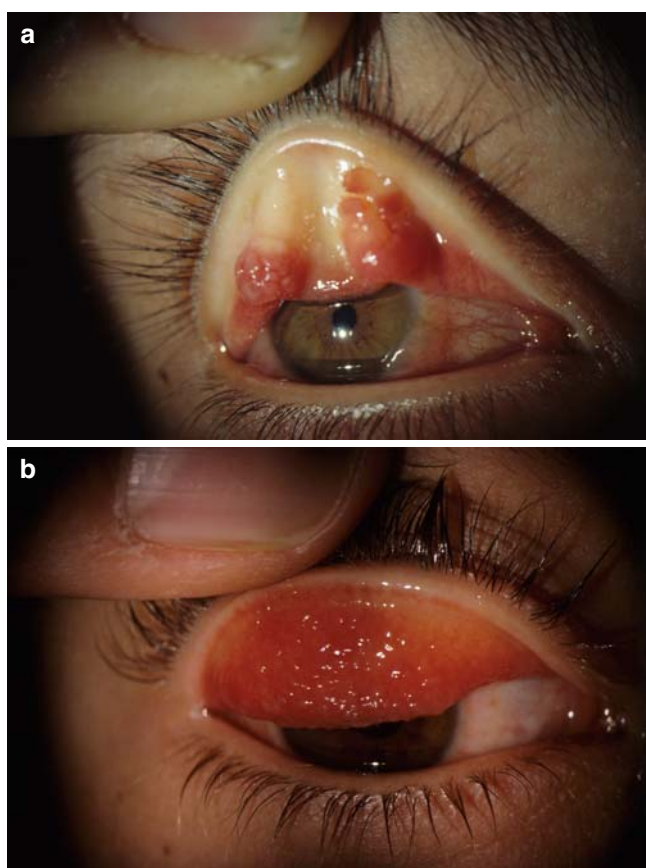


Fig.2.3.8 (a, b) Giant papillary conjunctivitis, generally seen in patients using contact lenses. Tarsal conjunctiva is sensitized to the allergen adhered to the outer surface of the lens. Due to continuous irritation, the upper tarsal conjunctiva develops giant papillae. The cornea is never involved. Discontinuing lens use together with some topical antiallergic eye drops such as sodium cromoglycate helps the situation (courtesy of Kiratli)



Fig.2.3.9 Allergic shiners, dark discoloration of the lower lids



Fig.2.3.10 Stuffed animals with artificial fur are major dust reservoirs and should not be kept in the room of an allergic child

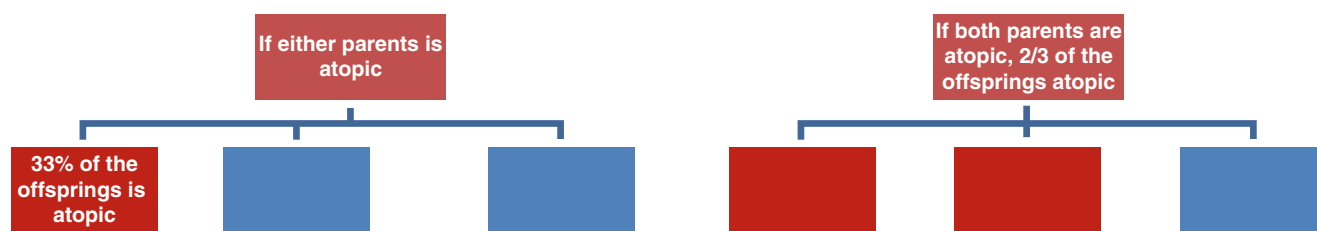


Fig.2.3.11 Inheritance of atopic diseases

Table 2.3.1 Pollen avoidance

Avoid going on picnics during the pollen season
Wear sunglasses outdoors
Stay inside in the late afternoon ^a
Keep the windows closed in the late afternoon ^a
Keep the windows closed in the car
Use pollen filters if possible

^aPollens rise with the heat during the day and come down as the air starts cooling during late afternoon. Therefore, in the late afternoon exposure is highest

Table 2.3.2 Dust mite allergen avoidance^a

Indoors
Avoid humidity
Avoid warm environment
Ventilate adequately
Reducing the burden of allergens
Avoid wall-to-wall carpeting; tile floors with small rugs are preferable
Remove dust reservoirs such as stuffed animals with artificial fur, soft toys, woollen blankets, old mattresses, silk flowers, mounted animals, books on open shelves, feather pillows or bedding, upholstered furniture
Use pillow covers and mattress covers impermeable to dust mites
Removing the allergens
Superfiltering or HEPA filter vacuum cleaners preferable (at least twice a week)
Clean with a damp cloth every week
Wash the laundry with water hotter than 60°C every week; if possible expose to sunlight

^aA general understanding of allergy and treatment is important. Fulfilling these measures only partially may not be enough to prevent the symptoms. For example, killing the mites with chemicals may not eradicate the allergen. It may persist for months or even years. Since the mites live in deep pores and attach to the fabric, deep vacuum cleaning is needed to remove the allergen

2.4

Nasal Vestibulitis and Nasal Furunculosis and Mucormycosis

Infection of the skin of the nasal vestibule is termed nasal vestibulitis. It may be secondary to constant rhinorrhea, nose picking, or viral infections such as herpes simplex and herpes zoster. Foreign bodies frequently cause vestibulitis in children due to purulent discharge. Nasal furunculosis is *Staphylococcus aureus* infection of the hair follicles. Nose picking is a frequent cause of furunculosis. Topical and if necessary systemic antibiotics are prescribed. The patient should be instructed not to squeeze out pus from this area. Since the veins draining this area are valveless and directly join the cavernous sinus, there is a potential risk of spreading infection to the cavernous sinus via these facial veins. Eczema may also mimic vestibulitis. In these cases steroid base ointment may help the patient. In persistent vestibulitis, neoplastic diseases such as basal cell or squamous cell carcinoma should be kept in mind.



Fig. 2.4.1 Nasal vestibulitis on the left side. Note the slight edema and hyperemia as well as excoriation of the skin on the left side



Fig. 2.4.2 If not treated, the infection in the nasal vestibule may spread to the upper lip. The upper lip on the right side appears hyperemic and edematous

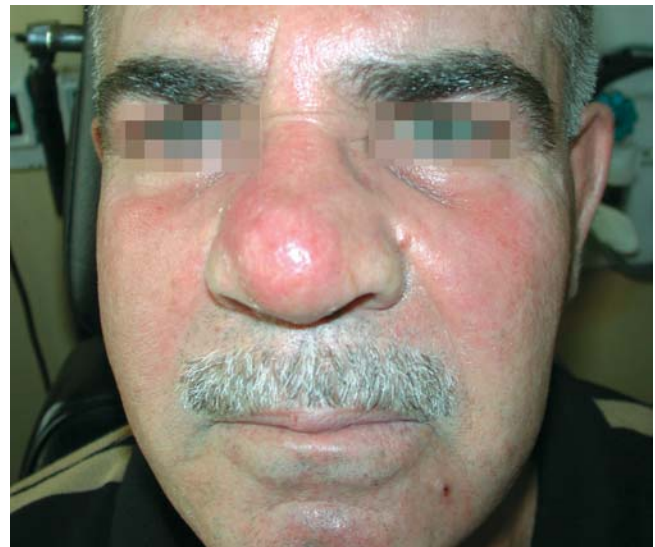


Fig. 2.4.3 Furunculosis in the nasal vestibule with spread of the infection to the nasal tip and dorsum



Fig. 2.4.4 Infection starting as nasal vestibulitis with spread of infection to the nasal dorsum and right periorbital area

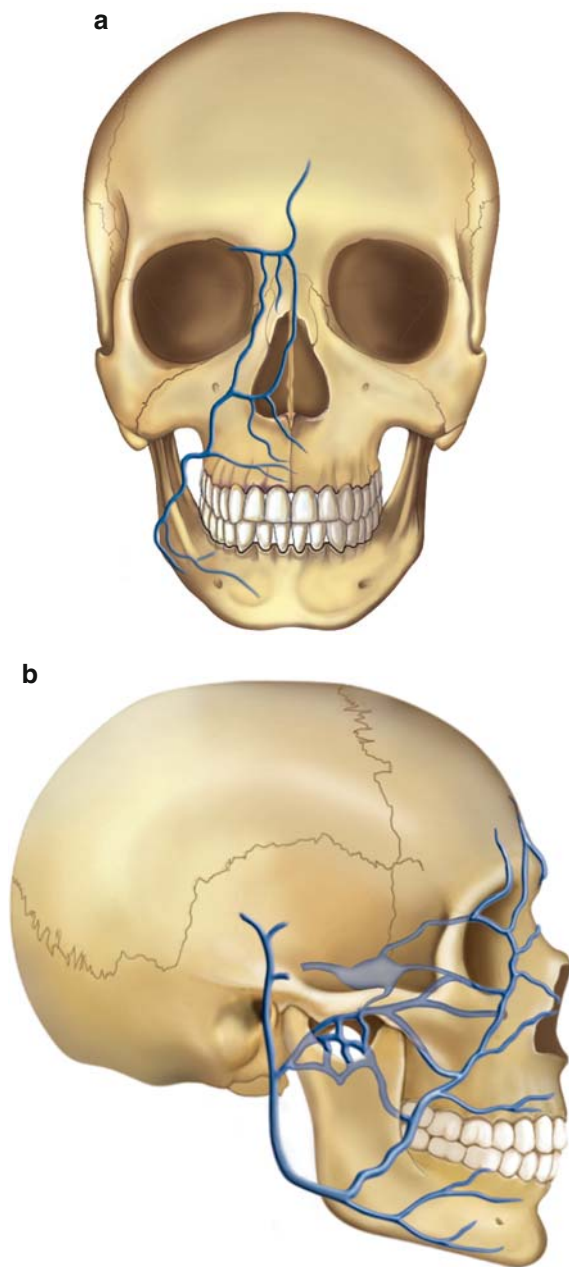


Fig. 2.4.5 Venous drainage of the nose. (a) frontal view, (b) Lateral view. Since the veins draining this area are valveless and directly join the cavernous sinus, there is a potential risk of spreading infection to the cavernous sinus via these facial veins. This area of the nose is termed the danger triangle. Squeezing the pus from this area should be avoided



Fig. 2.4.6 Constant rhinorrhea and the need to wipe the nose due to allergic rhinitis have resulted in vestibulitis

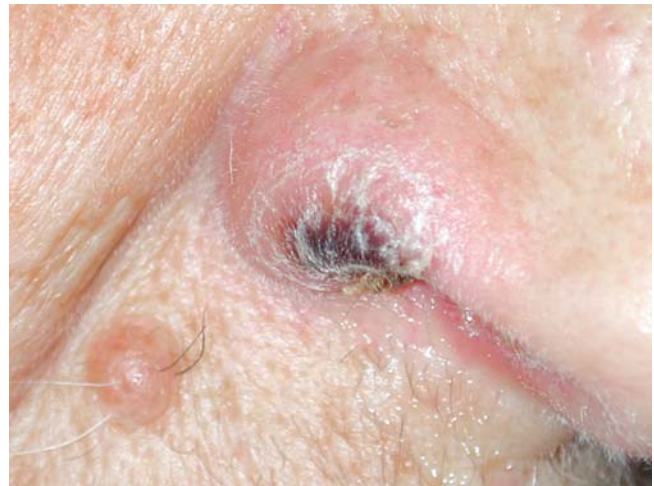


Fig. 2.4.7 Right alar rim. Basal cell carcinoma with slight hyperemia around it. In persistent vestibulitis, neoplastic diseases such as basal cell or squamous cell carcinoma should be kept in mind



Fig. 2.4.8 Hyperemia and edema in the columella and nasal tip mimicking severe vestibulitis secondary to squamous cell carcinoma infiltration. The neoplastic lesion is filling the left nasal passage

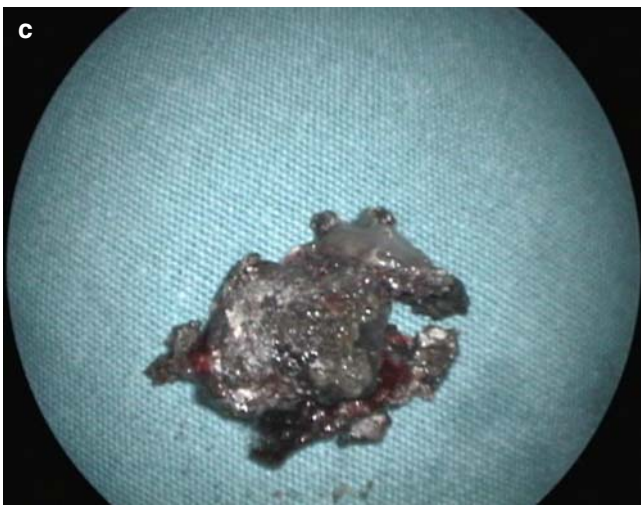


Fig. 2.4.9 Mucormycosis in a diabetic patient. Mucormycosis may infect different areas of the body, but the most frequent fatal form is the rhinocerebral form. (a) Necrotic areas on the face, (b) black necrotic areas in the nasal mucosa, (c) and after removal of necrotic area in the nose



Fig. 2.4.10 Mucormycosis in a child with leukemia. Gross tissue necrosis with a black eschar is characteristic of mucormycosis



Fig. 2.4.11 (a, b) Mucormycosis in an immunocompromised child. The disease progresses rapidly with extension of tissue necrosis out of the nose into the orbit and face. Local management requires wide debridement of necrotic tissue with a margin of normal-appearing tissue (Courtesy of TESAV)

2.5

Sinusitis

According to the duration of the disease, sinusitis is divided into two categories: acute and chronic.

Acute rhinosinusitis (ARS) is defined as the sudden onset of symptoms lasting less than 12 weeks (with symptom free intervals-complete resolution of symptoms, if the problem is recurrent). ARS can occur once or more than once in a defined time period. This is usually expressed as episodes/year, but

there must be complete resolution of symptoms between episodes for it to constitute recurrent ARS.

Chronic rhinosinusitis (CRS) is defined as disease lasting more than 12 weeks without complete resolution of symptoms. CRS may also be subject to exacerbations.

The presence of polypoid degeneration in the maxillary sinus deserves special attention. If the polyp is on the floor of the antrum, dental disease should be suspected. If the polyp is on the roof of the antrum, carcinoma should be ruled out in elderly patients. Any evidence of bone erosion should raise the possibility of carcinoma. In patients over 40 years of age, the possibility of carcinoma should always be kept in mind and intrasinus exploration should be performed if necessary.

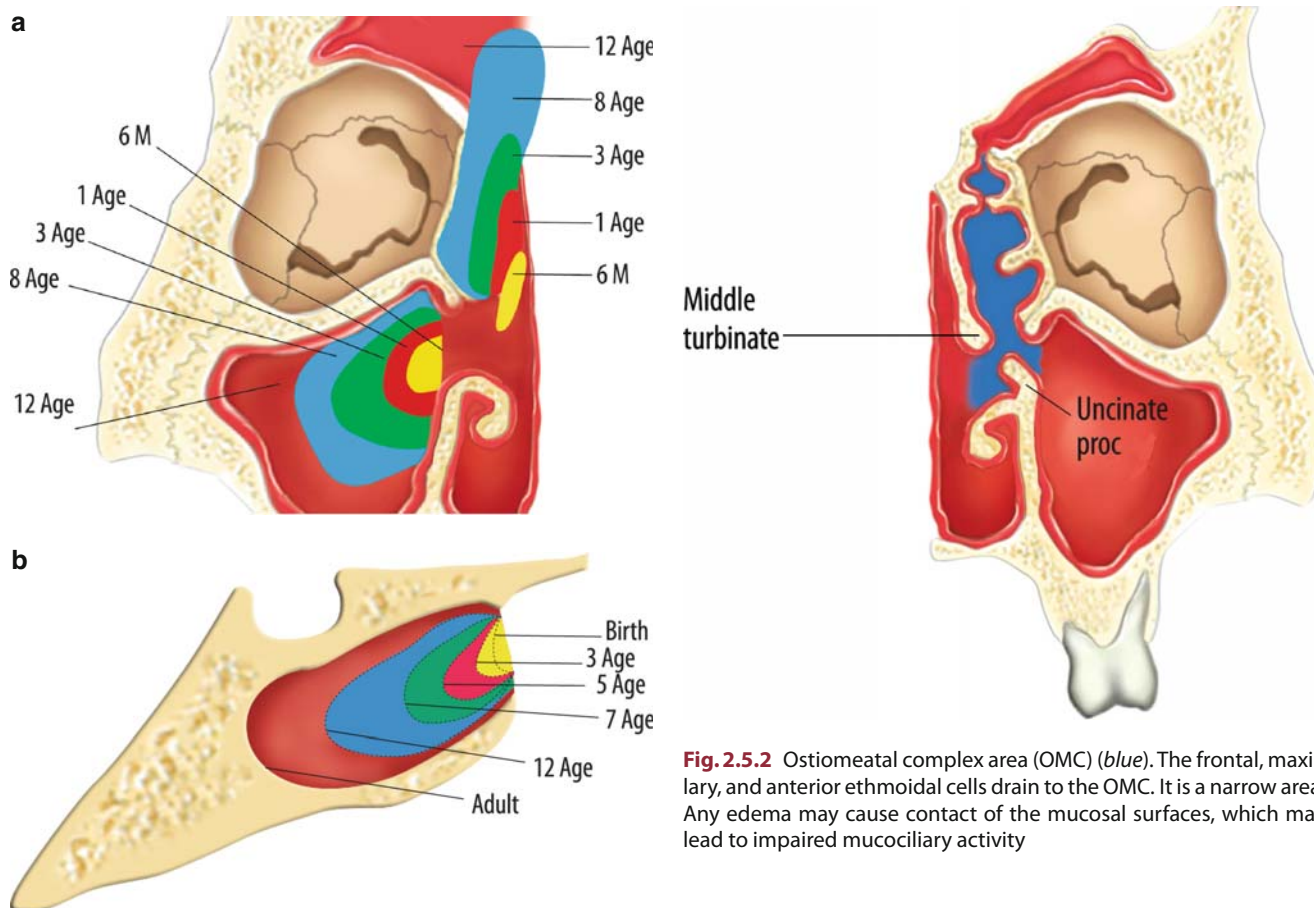


Fig. 2.5.1 Developmental anatomy of the ethmoidal, frontal, and maxillary sinuses (a) and sphenoidal sinus (b). The ethmoidal and maxillary sinuses are present at birth. The sphenoidal sinus is in the form of a small invagination and develops later. The frontal sinus develops from the anterior ethmoidal cells and moves from an infraorbital position to a supraorbital position, starting to develop at the age of 7. Yellow, 6 months; red, 1 year; green, 3 years; blue, 8 years; maroon, 12 years of age, adult size

Fig. 2.5.2 Ostiomeatal complex area (OMC) (blue). The frontal, maxillary, and anterior ethmoidal cells drain to the OMC. It is a narrow area. Any edema may cause contact of the mucosal surfaces, which may lead to impaired mucociliary activity

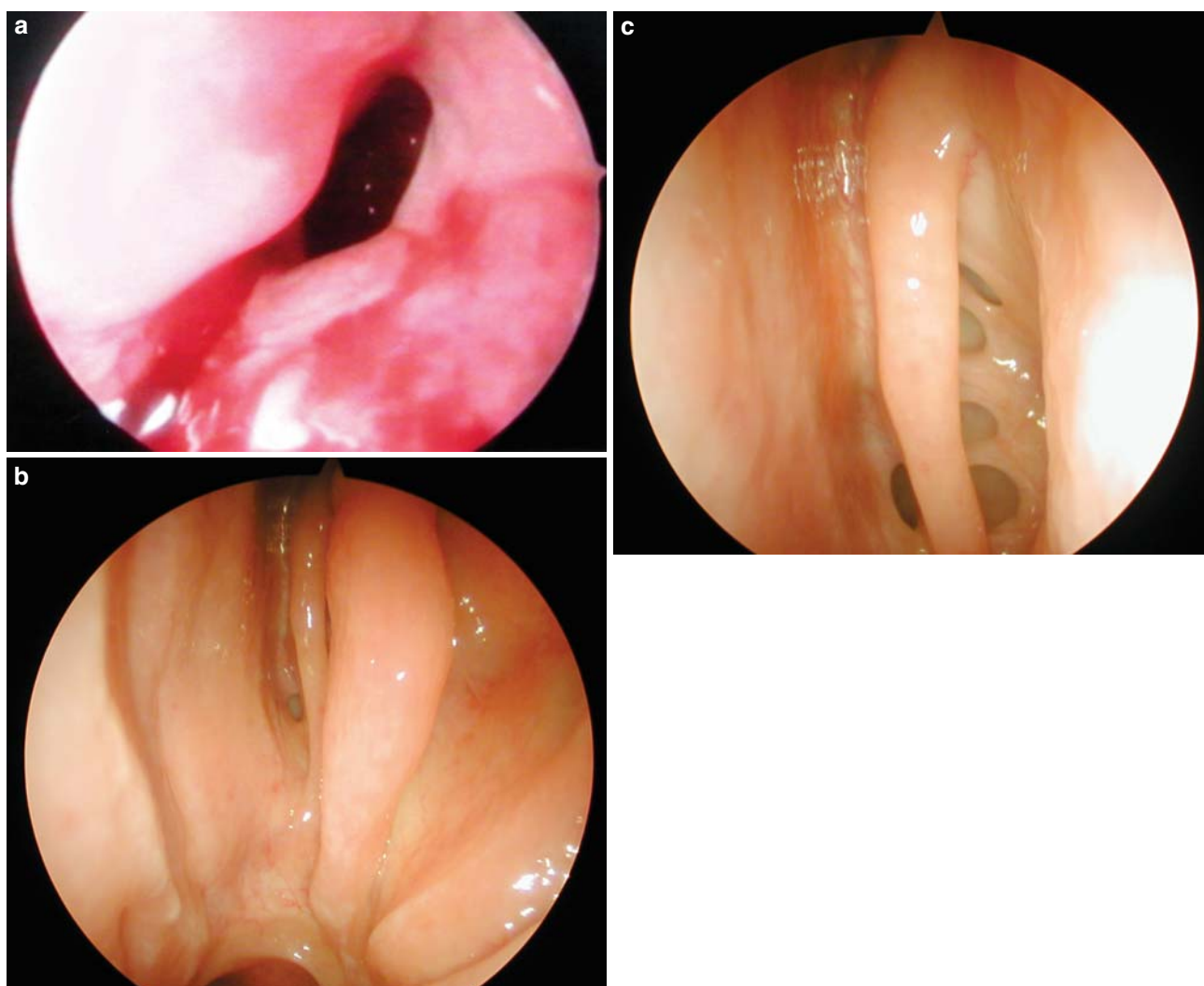


Fig. 2.5.3 (a) Maxillary sinus ostium from nasal side, (b) OMC lateral to the middle turbinate. Superior and posterior to the middle turbinate, the superior turbinate and sphenoid sinus ostium are seen;

(c) superior turbinate. Lateral to the superior turbinate are the posterior ethmoidal cells, and medial to the superior turbinate is the sphenoid sinus ostium

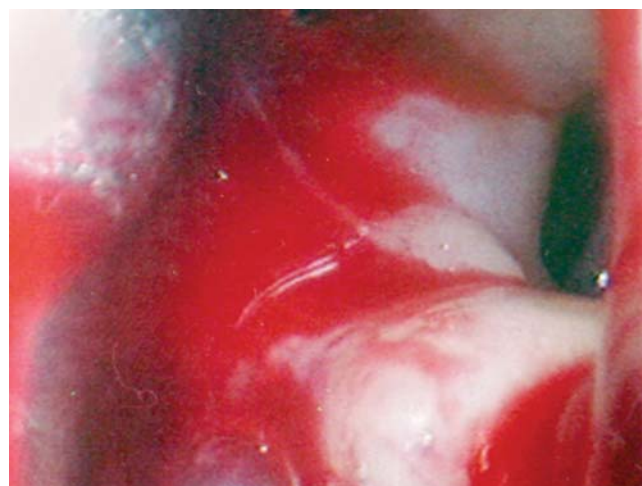


Fig. 2.5.4 Maxillary sinus ostium from the maxillary sinus side. Mucociliary activity is toward the ostium

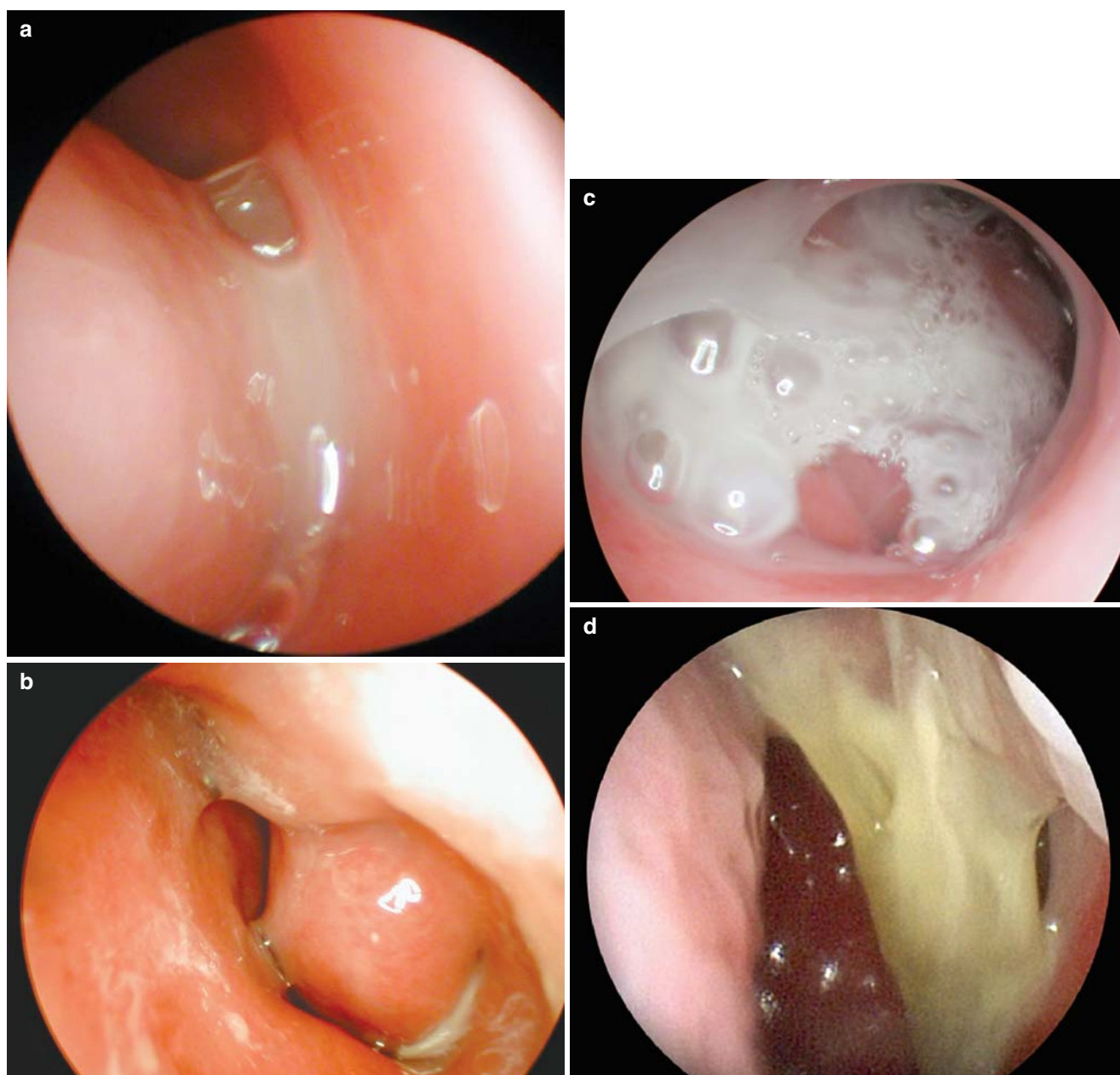


Fig. 2.5.5 Nasal discharge. (a) Muroid drainage after common cold; (b) purulent drainage in the inferior meatus; (c) postnasal purulent drainage; (d) allergic mucin: viscid, thick yellow-green drainage, generally eosinophilic



Fig. 2.5.6 Waters view showing right acute maxillary sinusitis. There is an air–fluid level in the right maxillary sinus



Fig. 2.5.7 Transillumination of the frontal sinus. In frontal sinusitis the frontal sinus fails to transilluminate

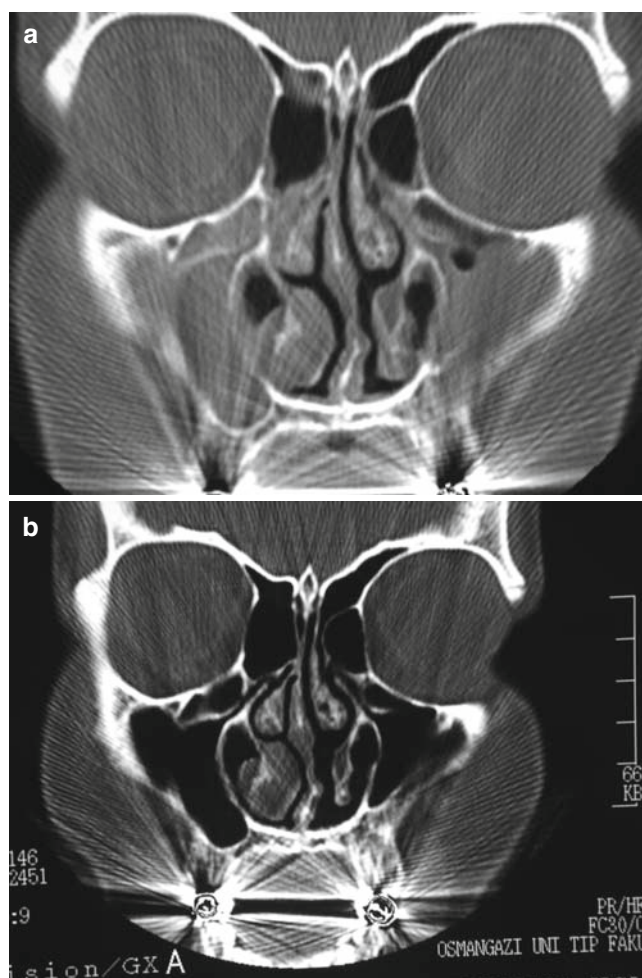


Fig. 2.5.8 (a) Coronal CT shows bilateral maxillary sinusitis; (b) 15 days after starting medical treatment the sinuses appear to be normal

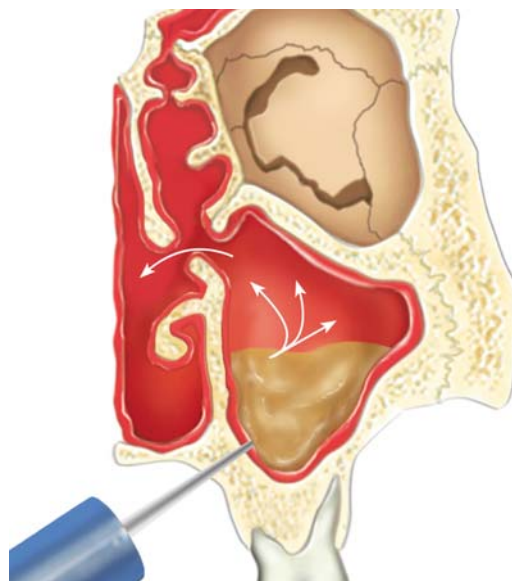


Fig. 2.5.9 Maxillary sinus irrigation. An opening is created via the inferior meatus between the nose and maxillary sinus or via canine fossa

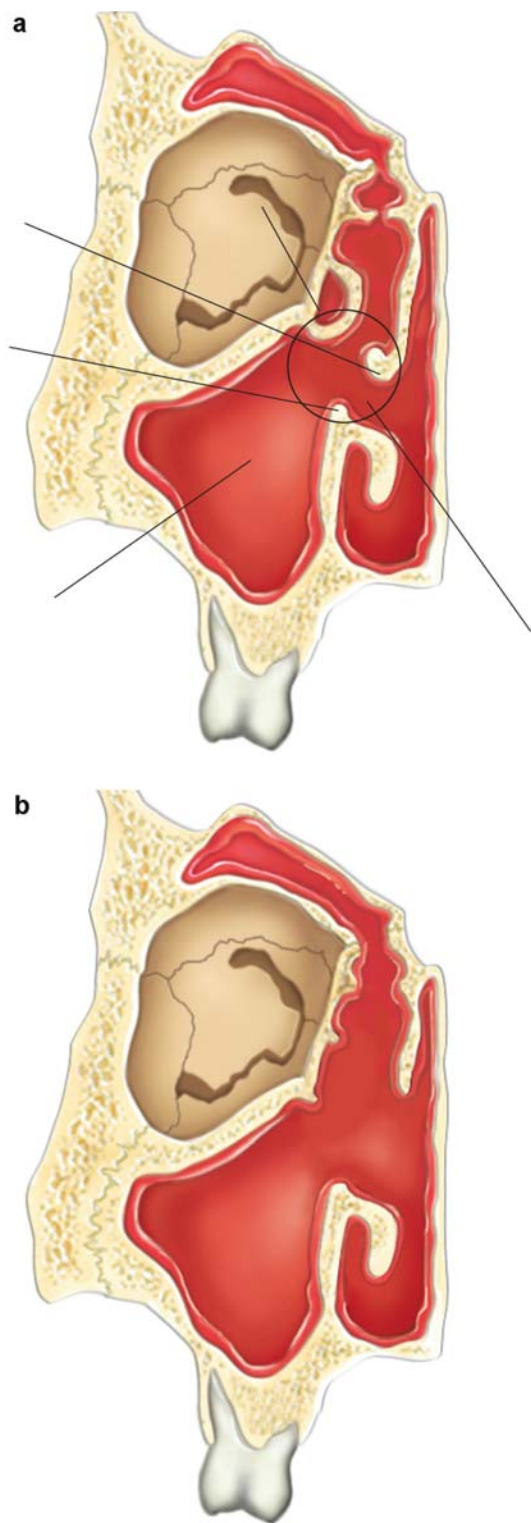


Fig. 2.5.10 The purpose of endoscopic sinus surgery is to restore ventilation and drainage of the paranasal sinuses. **(a)** Preoperative and **(b)** postoperative view

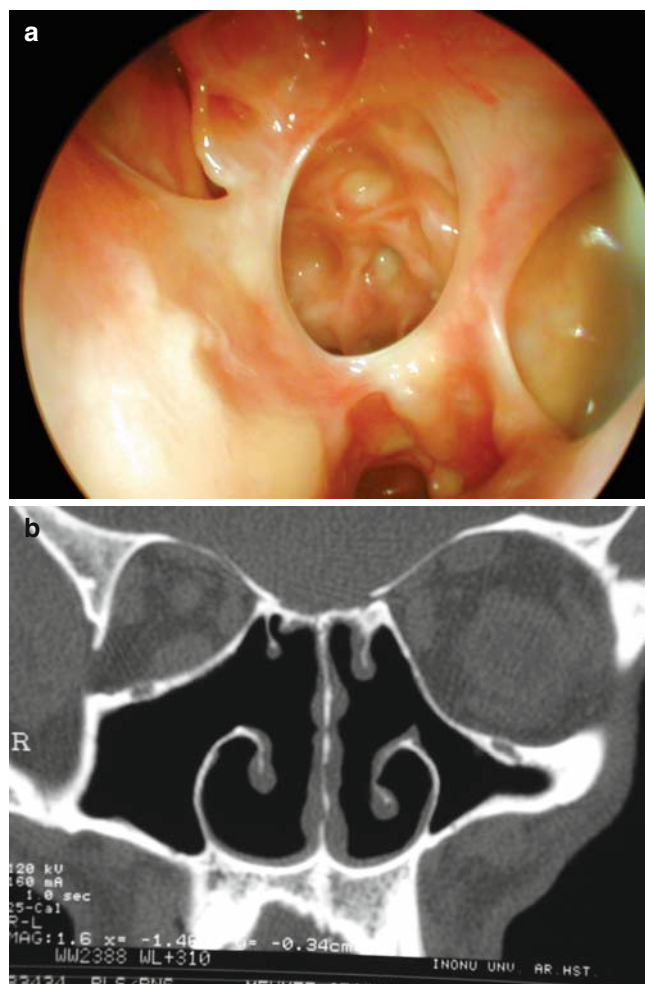


Fig. 2.5.11 **(a)** Epithelized sinuses and normal mucosa of the sinuses after endoscopic sinus surgery. **(b)** Coronal CT after surgery shows that all sinuses are clean and ostia are open

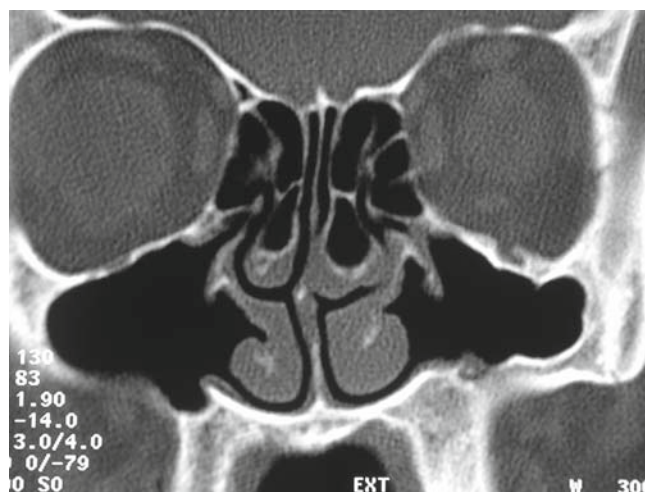


Fig. 2.5.12 Coronal paranasal sinus CT showing previously performed bilateral Caldwell-Luc operation. Note that both nasotracheal windows in the inferior meatus are widely open

Fig. 2.5.13 Different areas in the nasal cavity may cause referred pain in various regions of the head

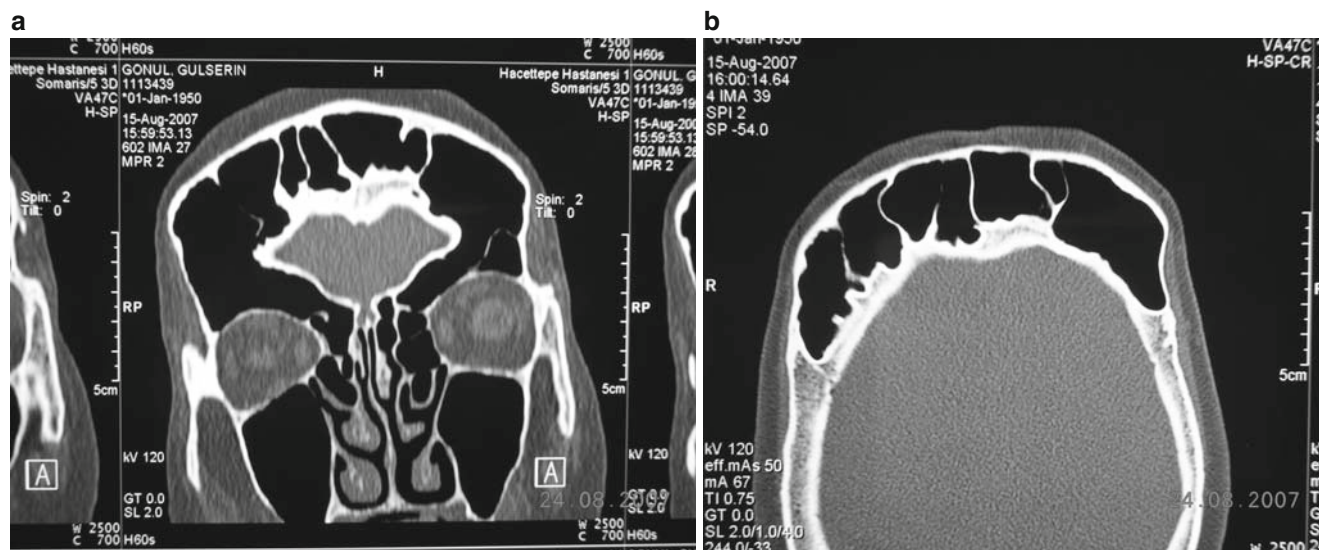
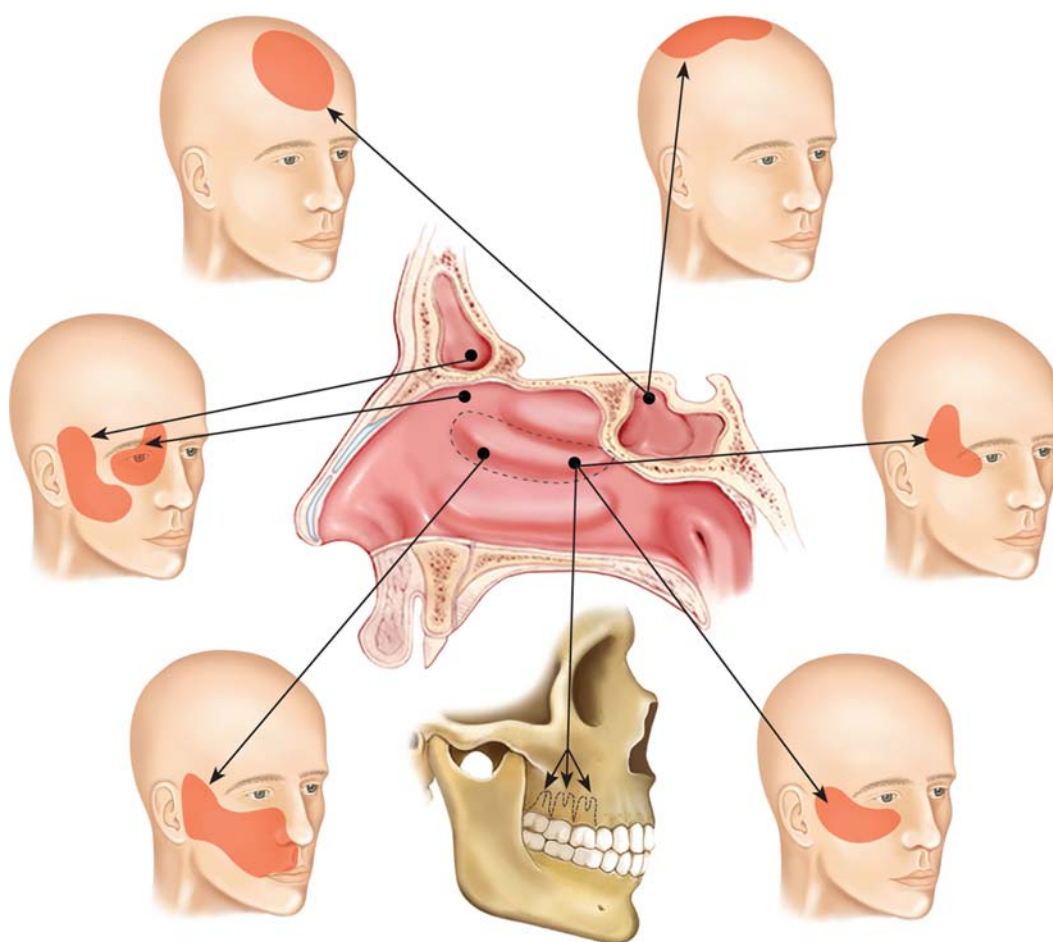


Fig. 2.5.14 (a, b) Overpneumatized frontal sinuses may cause annoying headaches due to negative pressure in the sinus in the frontal area. The pain is generally described as dull

Table 2.5.1 Why is the ostiomeatal complex (OMC) so important in sinusitis?

It is the most commonly diseased site
It is poorly visualized with anterior rhinoscopy
It cannot be evaluated with conventional X-rays
It is a very narrow area, and minor swellings cause obstruction
Symptoms are mild and overshadowed

Table 2.5.2 Sinus aspiration and irrigation indications

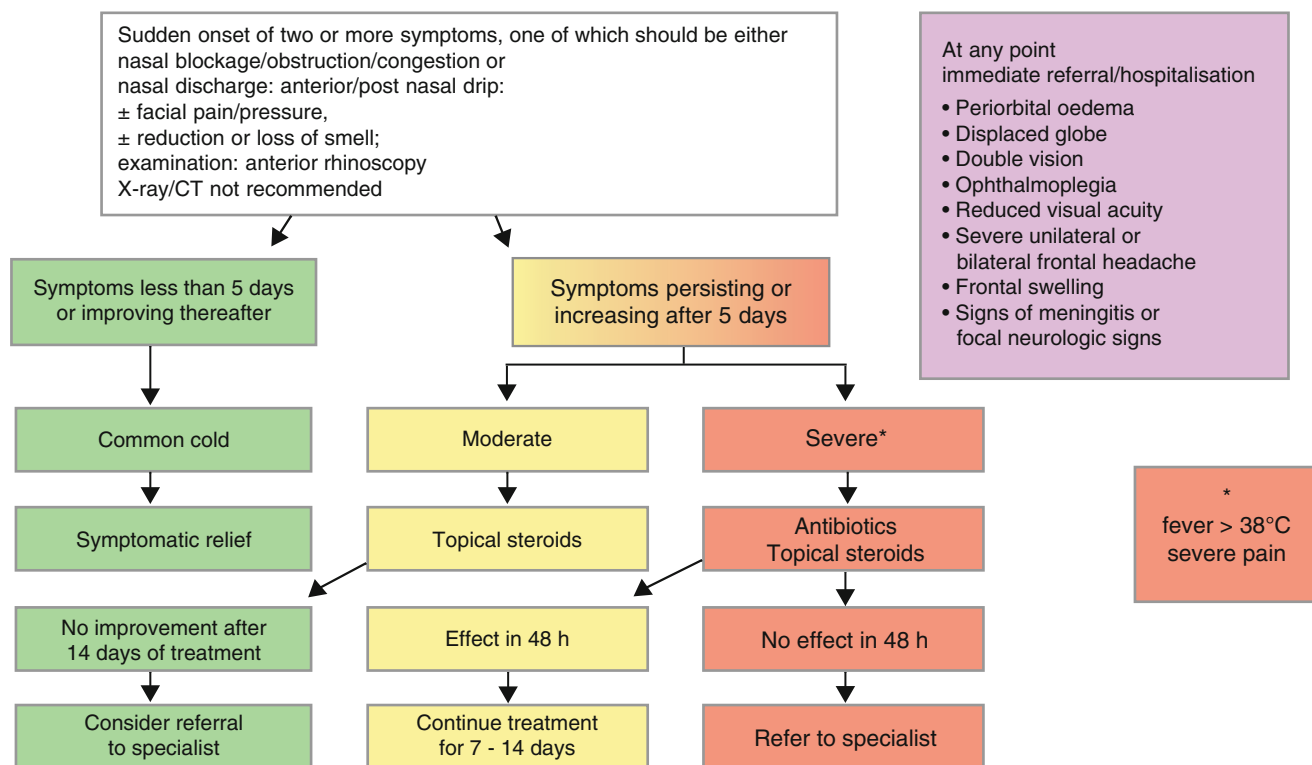
Clinical nonresponse to adequate conventional therapy
Immunocompromised patient
Severe symptoms of facial pain
Impending or presenting complications (intraorbital or intracranial)

Table 2.5.3 Sinusitis: host factors

Septal deformity: inhibits drainage of sinuses into the middle meatus
Molar tooth abscess: leads to unilateral maxillary sinusitis
Immunocompromised patients: leukemia – chemotherapy – diabetes – AIDS
Aspirin sensitivity
Intranasal foreign body

Table 2.5.4 Patients who should be referred to ENT surgeon

All frontal or sphenoidal sinuses unresponsive to medical therapy
All immunocompromised patients
All patients with complications of sinus disease
Chronic sinusitis unresponsive to medical management
Chronic sinusitis in children (consider adenoidectomy)

Table 2.5.5 Treatment scheme for primary care for adults with acute rhinosinusitis [Adapted from European Position Paper in Rhinosinusitis and Nasal Polyposis, Suppl 20, 2007]

2.6

Complications of Sinusitis

Although the incidence of complications of sinusitis decreased remarkably after the introduction of antibiotics, these complications may still be life threatening. Complications of sinusitis can be classified as local, orbital, and intracranial.

The most popular example of a local complication is frontal bone osteomyelitis. Frontal sinusitis may cause osteomyelitis of the anterior table of the frontal bone. The pus may collect between the bone and periosteum. This subperiosteal abscess is known as “Pott’s puffy tumor.” Orbital complications are very frequently due to ethmoidal sinusitis. Especially in children, the lamina papyracea is dehiscent and infection can easily spread to the orbit.

Table 2.6.1 Complications of sinusitis

Osteomyelitis
Frontal (Pott’s puffy tumor)
Intracranial
Epidural abscess
Subdural abscess
Cavernous sinus thrombosis
Meningitis
Brain abscess
Orbital
Inflammatory edema (periorbital cellulitis)
Subperiosteal abscess
Orbital cellulitis
Orbital abscess
Optic neuritis (cavernous sinus thrombophlebitis)

Table 2.6.2 Extension routes of infection in sinusitis^a

Osteitis (osteomyelitis in bones with bone marrow)
<i>Direct extension</i>
Congenital dehiscences
Fracture lines from previous head traumas
<i>Venous extension</i>
Retrograde thrombophlebitis between the sinus mucosal veins and orbital and dural veins; Septic emboli in diploic veins ^b

^aLymphatic spread plays no role in extension of sinus infections

^bThere are no valves in the veins connecting the orbit and sinuses (Breschet diploic veins) and this creates an easy route for extension of infection

Table 2.6.3 Stages of orbital complications of sinusitis

Periorbital cellulitis	Infection anterior to the orbital septum
Orbital cellulitis	Infection posterior to the orbital septum
Subperiosteal abscess	Pus collection beneath the periosteum and lamina papyracea
Orbital abscess	Pus collection in the orbit
Cavernous sinus thrombophlebitis	Extension of infection to cavernous sinus

Table 2.6.4 Orbital complications, eye mobility vs. vision

Complication	Extraocular muscle impairment	Visual acuity loss
Inflammatory edema	None	None
Subperiosteal abscess	Minimal, in early stages; may limit eye mobility significantly in big abscesses	None, very minimal in big abscesses
Orbital cellulitis	Minimal	Minimal
Orbital abscess	Complete	Severe
Cavernous sinus thrombosis	Complete, often bilateral	Severe, often bilateral



Fig. 2.6.1 Right preseptal cellulitis. It is seen particularly in children because of the dehiscences in the lamina papyracea. Sometimes when the ethmoid sinus is completely congested, periorbital swelling may occur due to obstruction of venous drainage

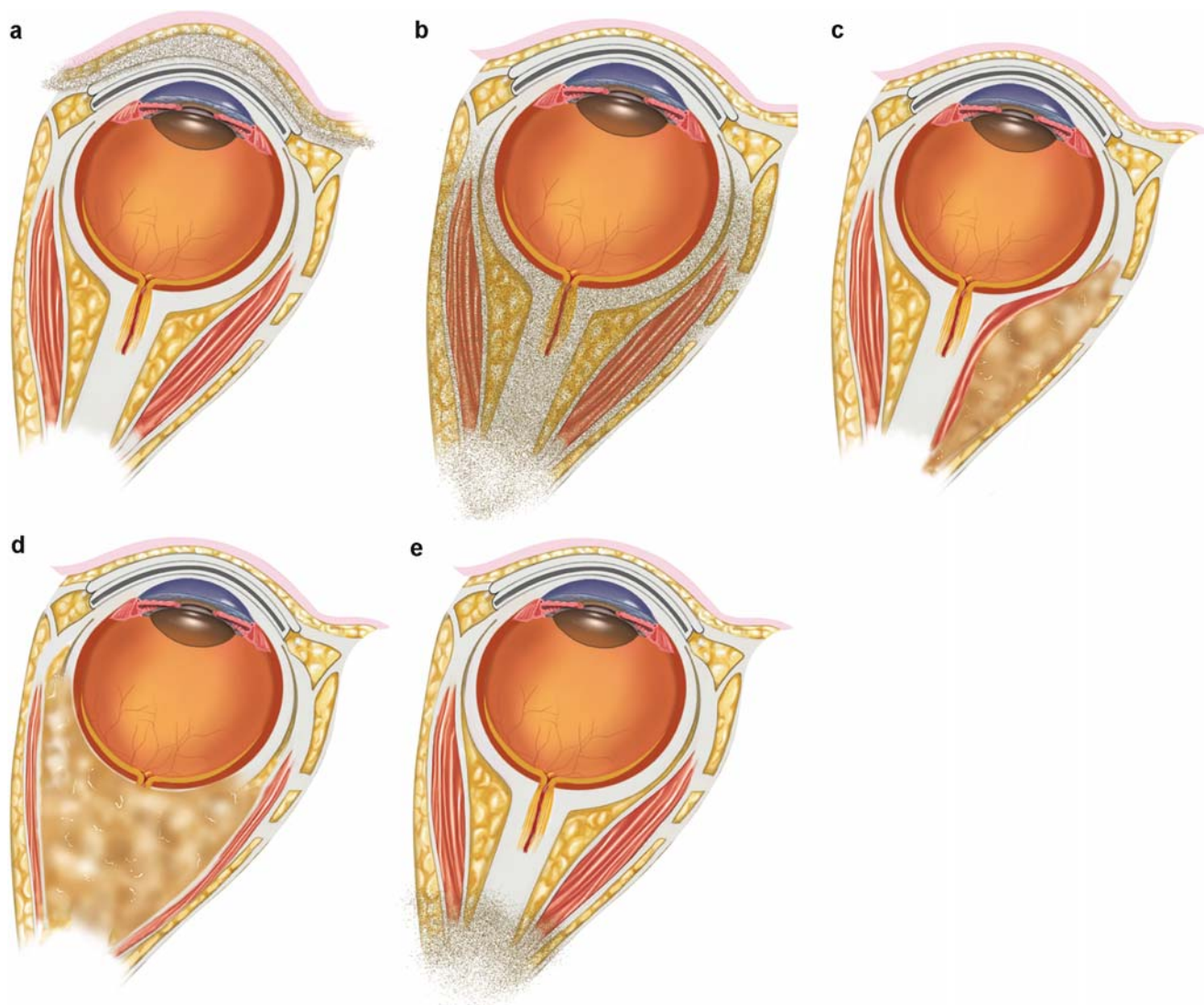


Fig. 2.6.2 Schematic drawing of orbital complications of sinusitis. **(a)** Preseptal cellulitis, **(b)** orbital cellulitis, **(c)** subperiosteal abscess, **(d)** orbital abscess, and **(e)** cavernous sinus thrombophlebitis

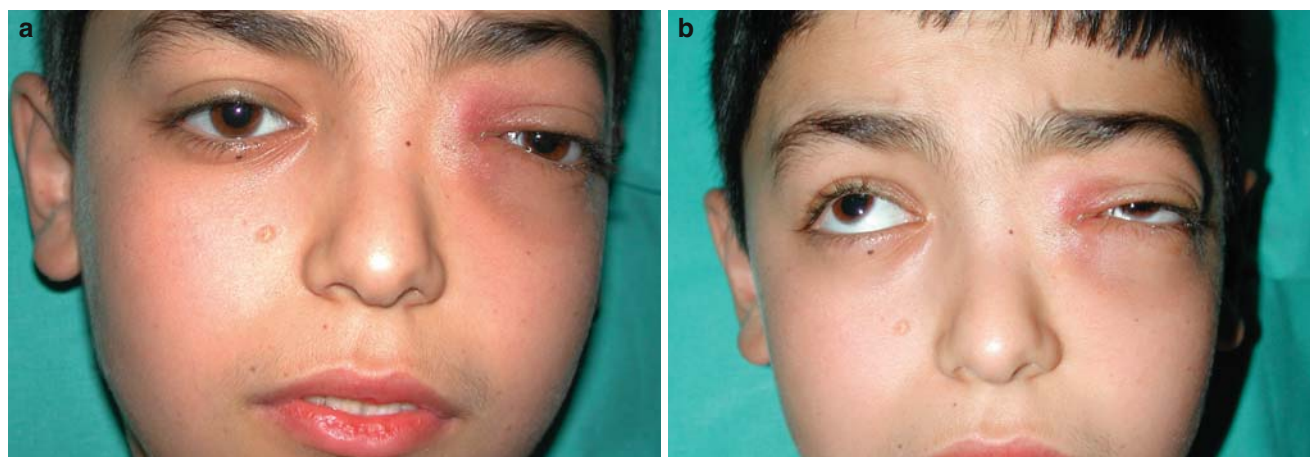


Fig. 2.6.3 Left-sided subperiosteal abscess. **(a)** The eye globe is pushed anteriorly and is displaced laterally and inferiorly by the subperiosteal abscess. The patient's upper lid is swollen, and **(b)** he is unable to elevate his left upper eyelid. His eye movements are limited in the upward and medial gaze. **(c)** Axial CT scan shows complete

opacification of the left ethmoid cells. There is a large subperiosteal abscess lateral to the lamina papyracea. Very small bony dehiscences can be noted in the lamina papyracea; **(d)** axial and **(e)** coronal MR images demonstrate complete opacification of ethmoid sinuses and large subperiosteal abscess

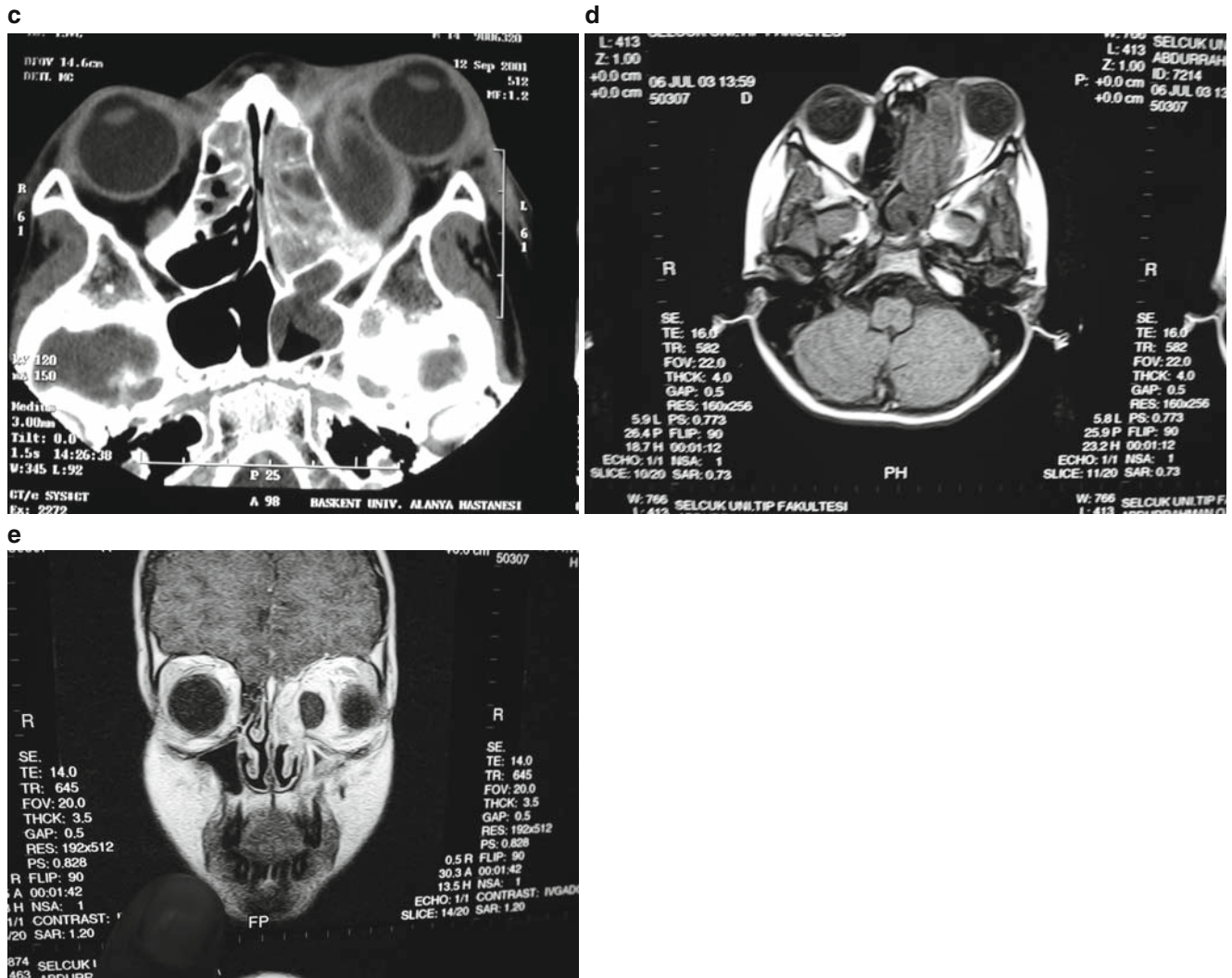


Fig. 2.6.3 (continued)



Fig. 2.6.4 Right orbital abscess due to ethmoiditis. Infection from the ethmoid sinuses may spread very easily via small dehiscences in the lamina papyracea into the orbit. External drainage may be required

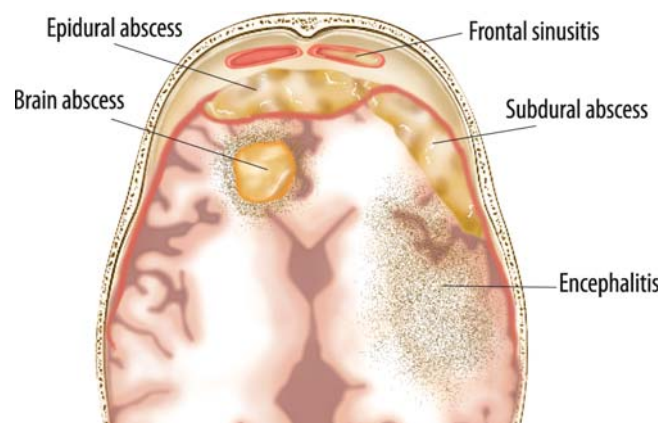


Fig. 2.6.5 Schematic representation of intracranial complications of frontal sinusitis. Purulent material may collect between the bone and dura (epidural abscess) or between the dura and the brain (subdural abscess) or in the brain (brain abscess)

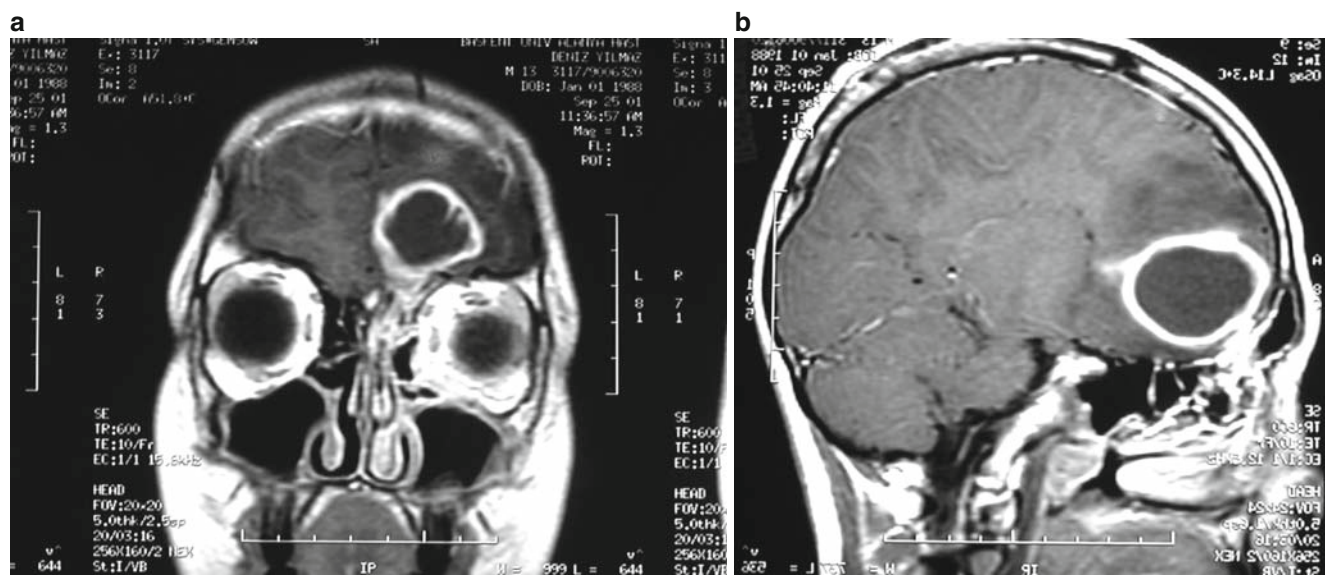


Fig. 2.6.6 Brain abscess. (a) Coronal and (b) sagittal MR images

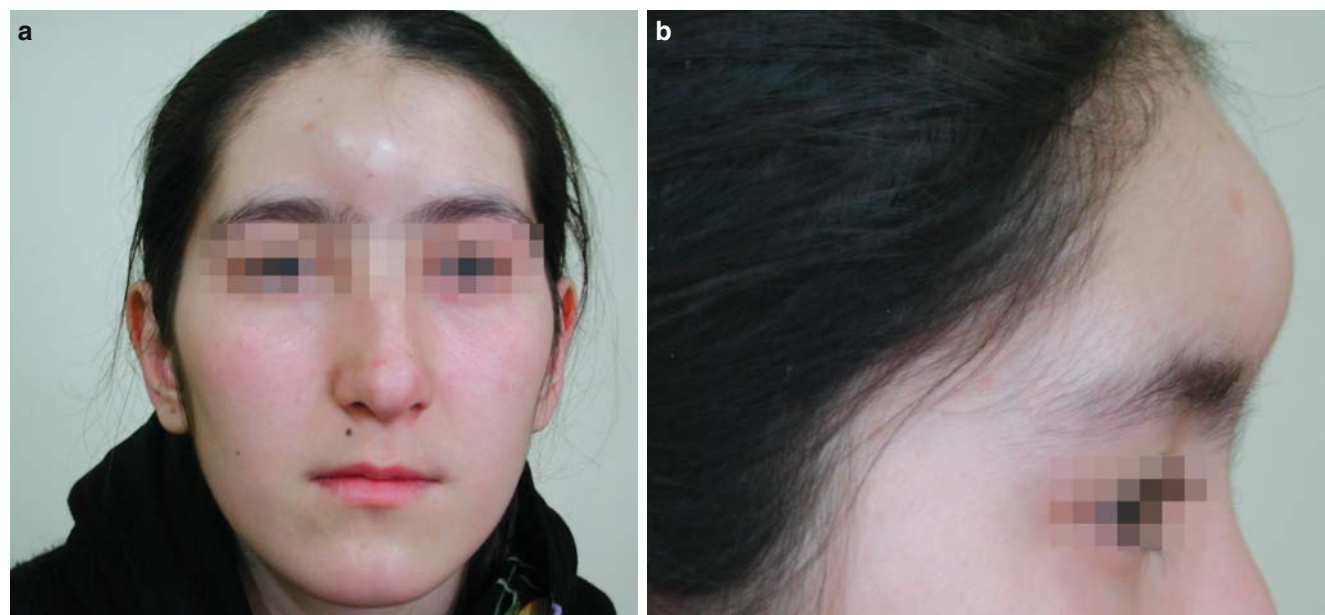


Fig. 2.6.7 Pott's puffy tumor. Following acute frontal sinusitis, the patient developed subperiosteal abscess in the forehead area. The forehead is swollen, tender, and fluctuant. (a) Frontal view, (b) lateral view, (c, d) axial MR images, (e) and sagittal reconstruction (the frontal sinus is opaque). Subperiosteal abscess is seen under the soft tissues of the forehead

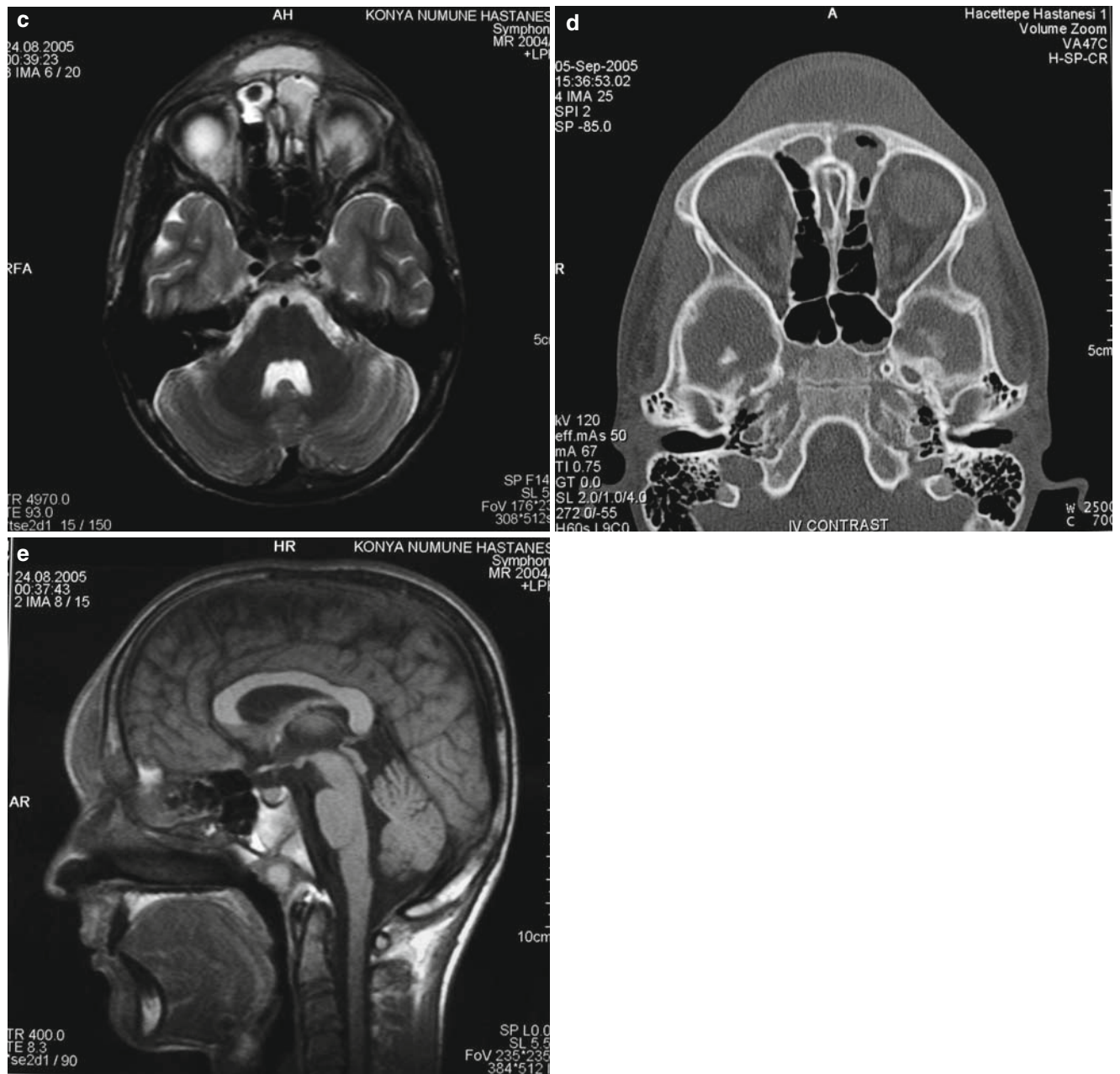


Fig. 2.6.7 (continued)

Fig. 2.6.8 (a, b) Axial and coronal CT scans of frontal sinus mucocoele. The ethmoid sinuses are completely opaque, and the posterior and superior wall of the frontal sinus is destroyed. Erosion of the roof of the orbit leads to orbital displacement inferiorly and laterally

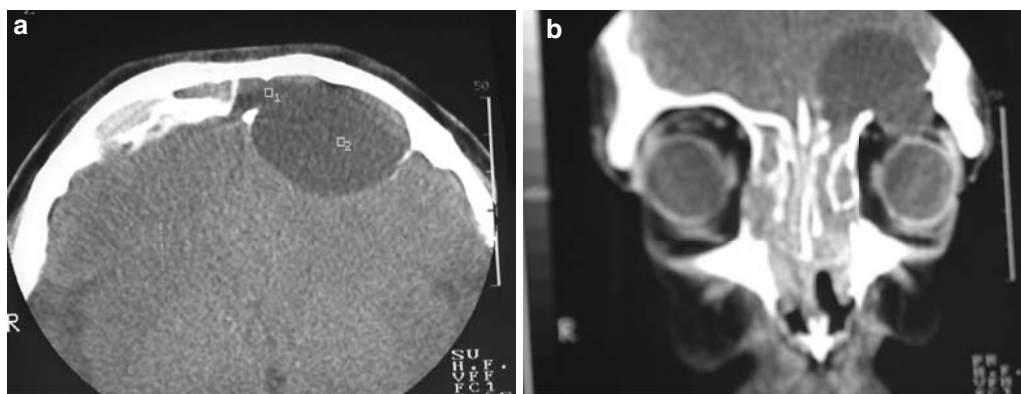


Fig. 2.6.9 Left-sided ophthalmoplegia. The eye globe does not move in any direction. **(a)** The left upper lid is ptotic; **(b)** left eye does not move in lateral direction; **(c)** left eye does not move in medial direction; **(d)** left eye does not move in inferior direction; **(e)** left eye does not move in superior direction. Superior orbital fissure syndrome is characterized by the involvement of the 3rd, 4th, 6th, and ophthalmic branch of the trigeminal nerve. Vision is normal. In orbital apex syndrome, the optic foramen is also involved and there is loss of vision due to optic nerve involvement in addition to the superior orbital fissure syndrome

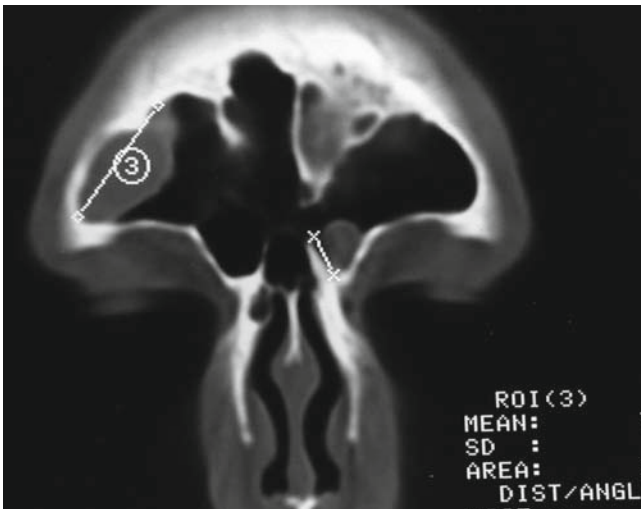


Fig. 2.6.10 Supraorbital frontal cell in the lateral wall of the frontal sinus. If they do not cause any symptoms, there is no need for surgery. To treat these disorders, osteoplastic frontal sinus operation is necessary



Fig. 2.6.12 Due to ethmoidofrontal mucocoele, the right eye is pushed laterally and inferiorly. The eye globes are not at the same level

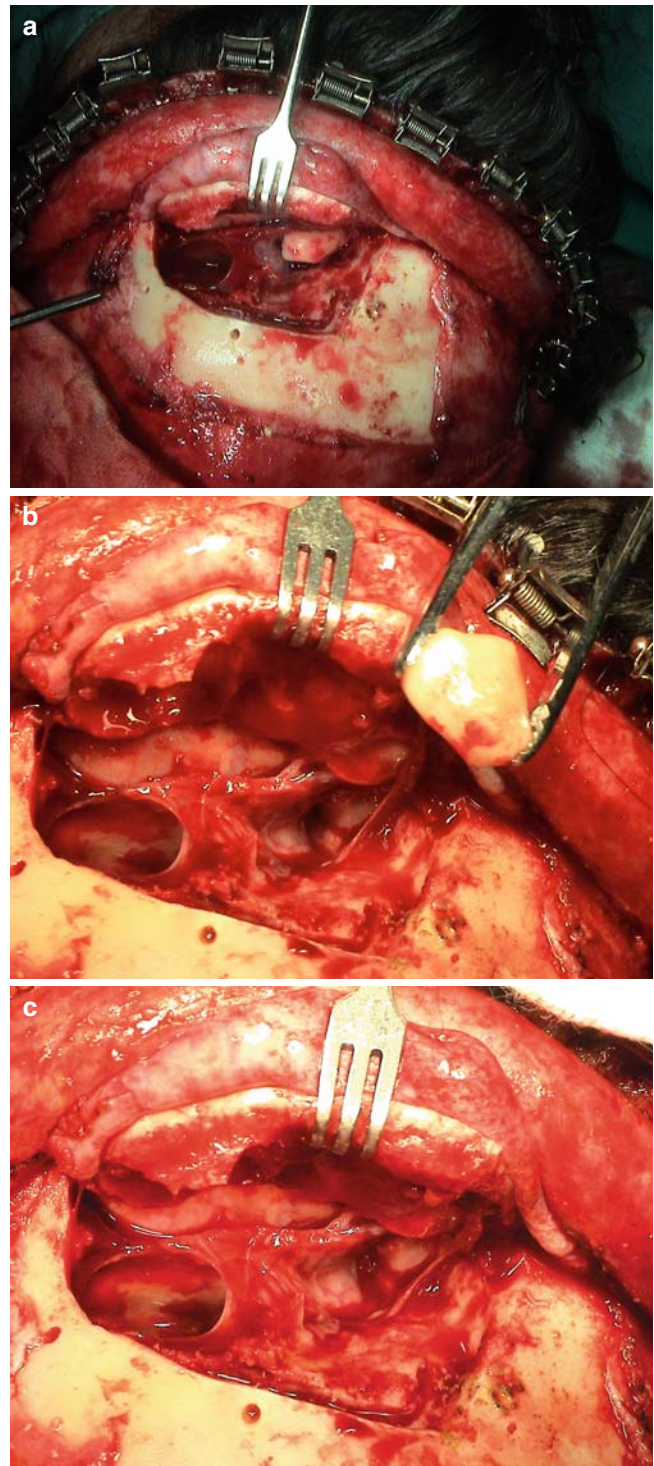


Fig. 2.6.11 (a–c) Osteoplastic frontal sinus operation in a patient with frontal sinus osteoma. The anterior table of the frontal sinus is elevated. The periosteum is not separated from the anterior table and is pedicled on the bone. After removing the osteoma and cleaning the pathology, the anterior table is placed back into the original position. Although this procedure is performed rarely, it provides a wide exposure in complicated diseases, trauma, tumor, or CSF fistula of the frontal sinus

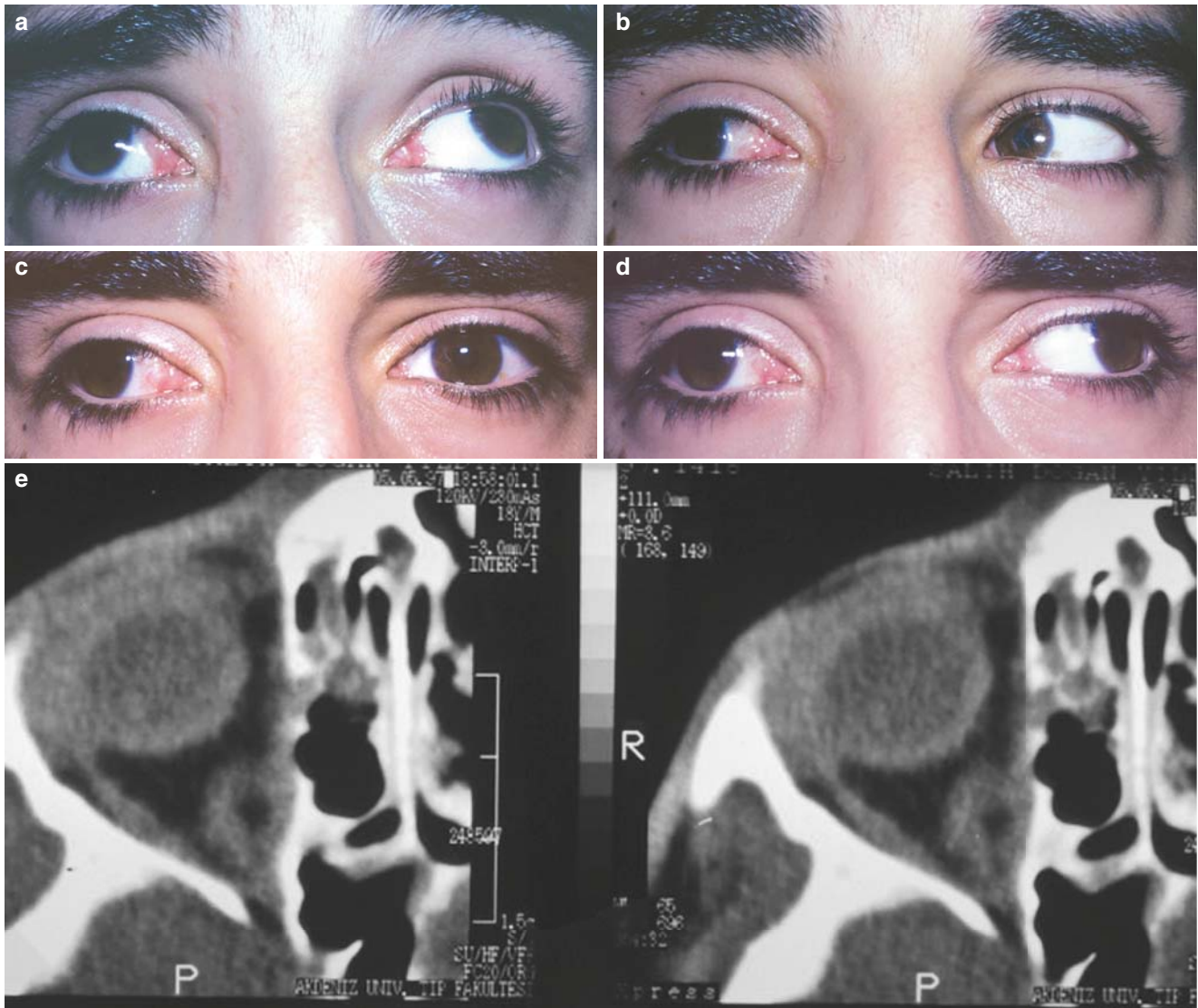


Fig. 2.6.13 Medial rectus muscle injury on the right side during endoscopic sinus surgery. **(a–d)** There is strabismus to the lateral side and impaired mobility of the eye. **(e)** Axial CT scans show the injury (courtesy of Şener)

2.7

Nasal Polyposis

Polyps are one of the most frequent causes of nasal obstruction. Polyps may be isolated or diffuse. The behavior of nasal polyps depends on the type of granulocytes. Eosinophils play a significant role in the classification of polyps. Polyps with significant eosinophilia behave differently from those with neutrophils. The majority of diffuse nasal polyposis is eosinophilic. Analgesic intolerance and asthma might accompany diffuse eosinophilic nasal polyposis in the later decades of a patient's life. Isolated polyps can originate from an anatomic structure such as the ethmoid bulla or uncinate process. If polyps originate from the mucosa inside the sinus, they are named according to the sinus. If the polyp originates from the maxillary sinus it is called an antrochoanal polyp, and if it originates from the sphenoid sinus it is called a sphenchoanal polyp.

Nasal polyps are unusual in children. If a child presents with nasal polyposis, the possibility of diseases such as cystic fibrosis or primary ciliary dyskinesia should be eliminated. If the nasal polyp is unilateral, nasal encephaloceles should be ruled out with MR imaging.

In diffuse nasal polyposis patients, the presence of asthma or analgesic intolerance should always be questioned. Due to the danger of intolerance which can develop later in their life, NSAIDs should not be prescribed to patients with diffuse eosinophilic nasal polyposis.

In adults, unilateral polyps should always raise the clinical suspicion of malignancy.

Table 2.7.1 Classification of nasal polyposis

Polyps
Inflammatory
Choanal/ isolated
Eosinophilic
Additional criteria
Acetylsalicylic acid intolerance
Asthma/COPD
Allergy
Associated diseases
Cystic fibrosis
Immune insufficiency (acquired/congenital)
Primary ciliary dyskinesia
Vasculitis, granulomatosis

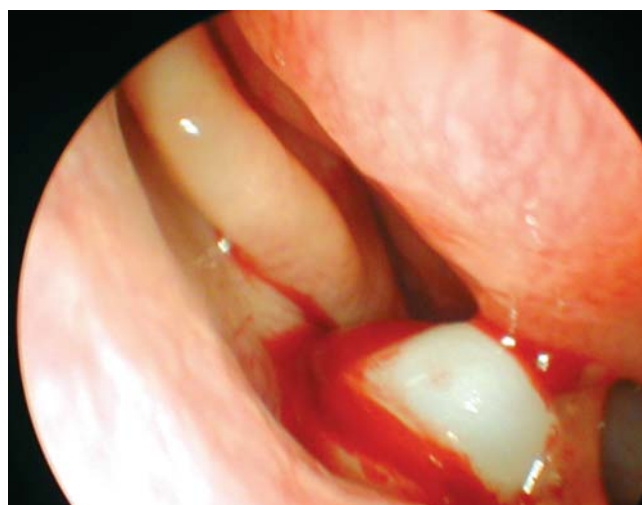


Fig. 2.7.1 Isolated polyp originating from the right middle meatus and extending anterior to the middle turbinate

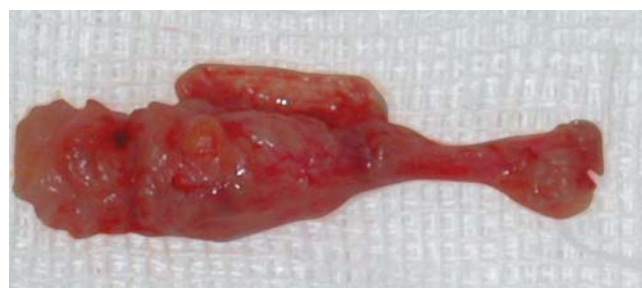


Fig. 2.7.2 Polyp originating from the uncinate process



Fig. 2.7.3 Right sphenchoanal polyp originating from the mucosa inside the sphenoid sinus, filling the sphenothmoidal recess, and extending to the choana

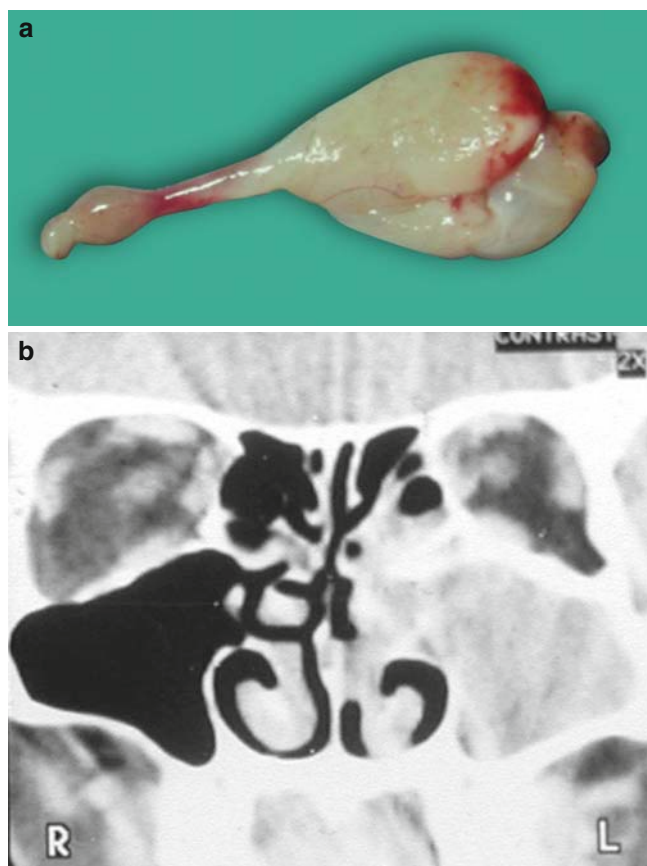


Fig. 2.7.4 (a) Antrochoanal polyp has three parts. The cystic portion is the part originating in the maxillary sinus. If this part is not removed, the polyp recurs. The neck is the portion of the polyp passing through the ostium. The main polyp mass is the part filling the nasal passage and causing obstruction. Although antrochoanal polyps are very often unilateral, they may cause bilateral nasal obstruction by extending and occluding the other choana. If the cystic part is not removed, the polyp recurs. (b) Coronal CT scan of antrochoanal polyp; the left maxillary sinus is opaque and the maxillary sinus ostium is widened. This widening of the maxillary sinus ostium is almost diagnostic for antrochoanal polyp

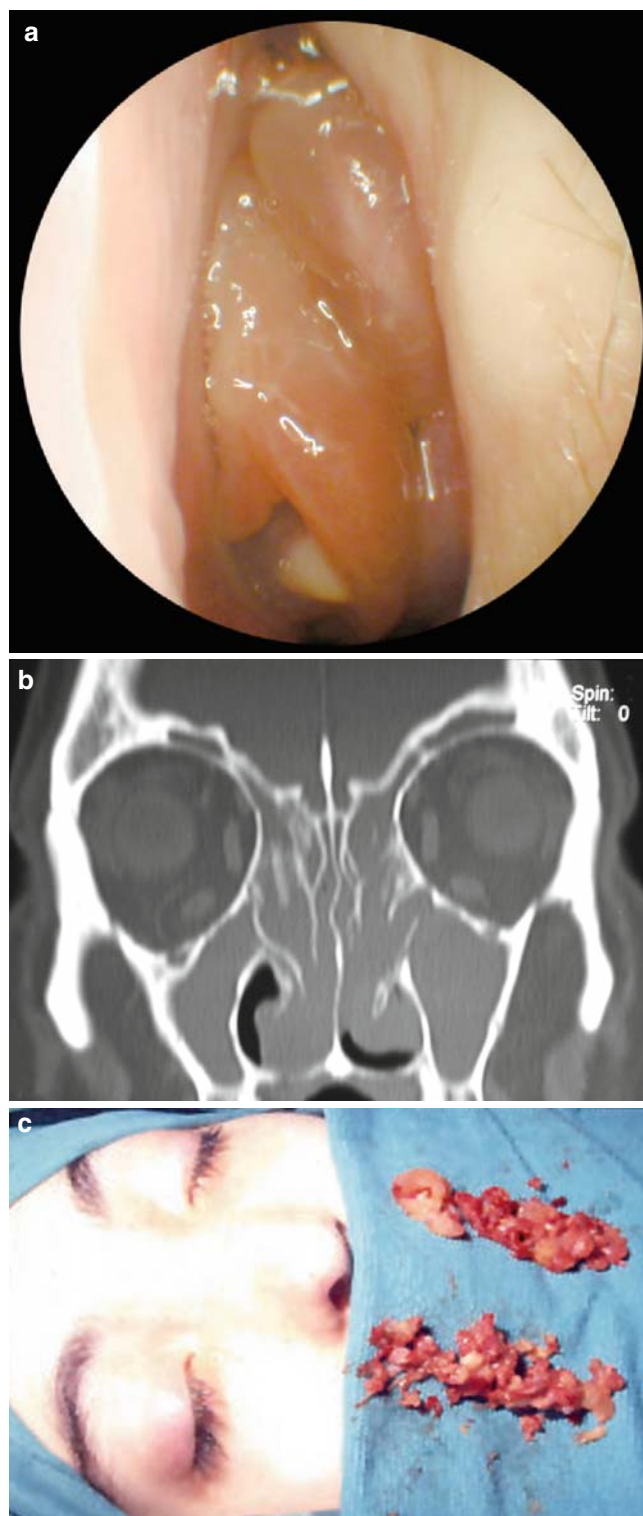


Fig. 2.7.5 (a) Diffuse eosinophilic nasal polyposis; the nasal passage is completely obstructed. Since the whole ethmoidal mucosa is polypoid there is no originating point. (b) Coronal CT scan shows that all sinuses are opaque. (c) Removal of the polyps



Fig. 2.7.6 Diffuse eosinophilic nasal polyposis completely obstructing the nasal passage

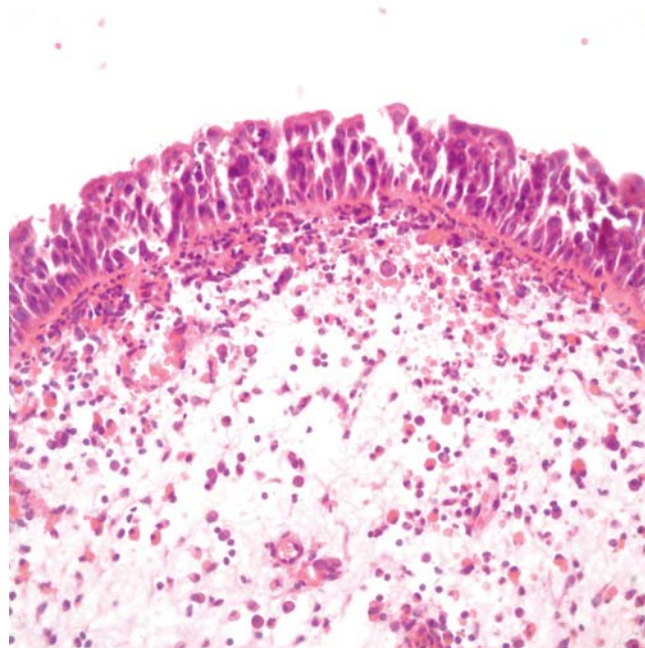


Fig. 2.7.8 Diffuse nasal polyposis. Diffuse eosinophilia is seen on histological examination (H&E)



Fig. 2.7.7 In eosinophilic diffuse nasal polyposis, the mucus is thick, viscid, and *yellow-green*. It contains many eosinophils. It is referred to as allergic mucin



Fig. 2.7.9 Fungal ball in sphenoid sinus

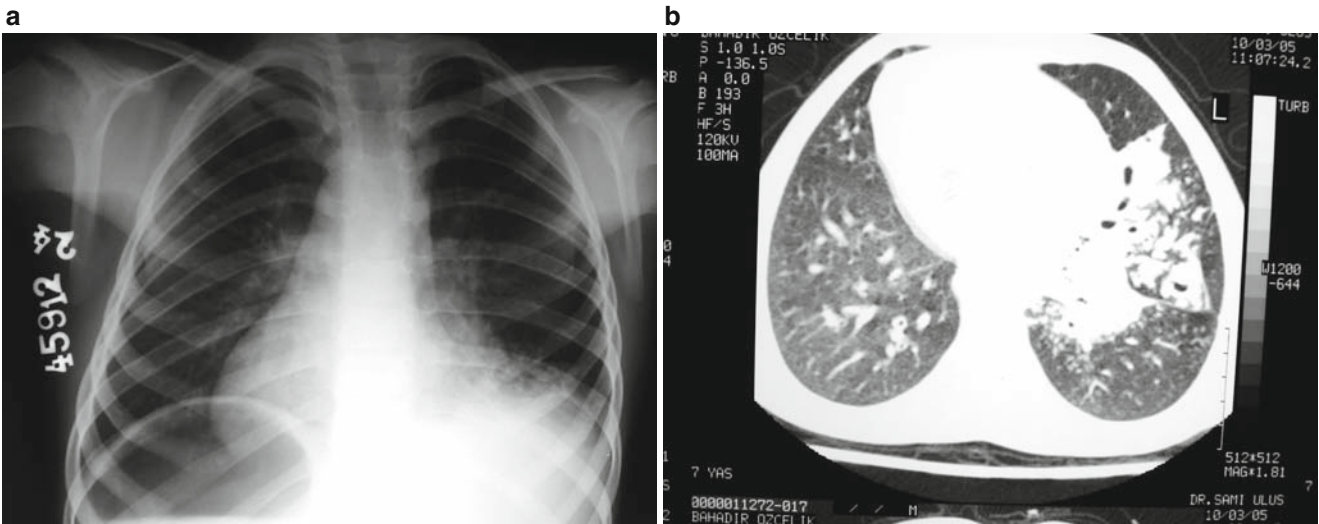


Fig.2.7.10 Kartagener syndrome. (a) Dextrocardia on chest X-ray; (b) diffuse bronchiectasia on axial CT scan



Fig.2.7.11 Nasal bone expansion due to extensive diffuse nasal polyposis in the younger patient. Rhinoplasty is needed to restore the appearance



Fig.2.7.12 Diffuse nasal polyposis completely filling the nasal passages. On the left side, nasal polyps protrude from the nostril. The patient did not agree to the operation because of her fear of anesthesia

Table 2.7.2 Treatment evidence and recommendations for postoperative treatment in adults with nasal polyps^a

Therapy	Level	Grade of recommendation	Relevance
Oral antibiotics: short term < 2 weeks	No data	D	Immediately postoperative, if pus was seen during operation
Oral antibiotics: long term > 12 weeks	Ib	A	Yes
Topical steroids after Functional endoscopic sinus surgery (FESS)	Ib (two studies one +, one -)	B	Yes
Topical steroids after polypectomy	Ib	A	Yes
Oral steroids	No data	D	Yes
Nasal douche	No data	D	Yes

After Table 13.6 of European Position Paper in Rhinosinusitis and Nasal Polyposis, Suppl 20, 2007. Reproduced with permission of *Rhinology*
^aSome of these studies also included patients with Chronic Rhinosinusitis (CRS) without nasal polyps

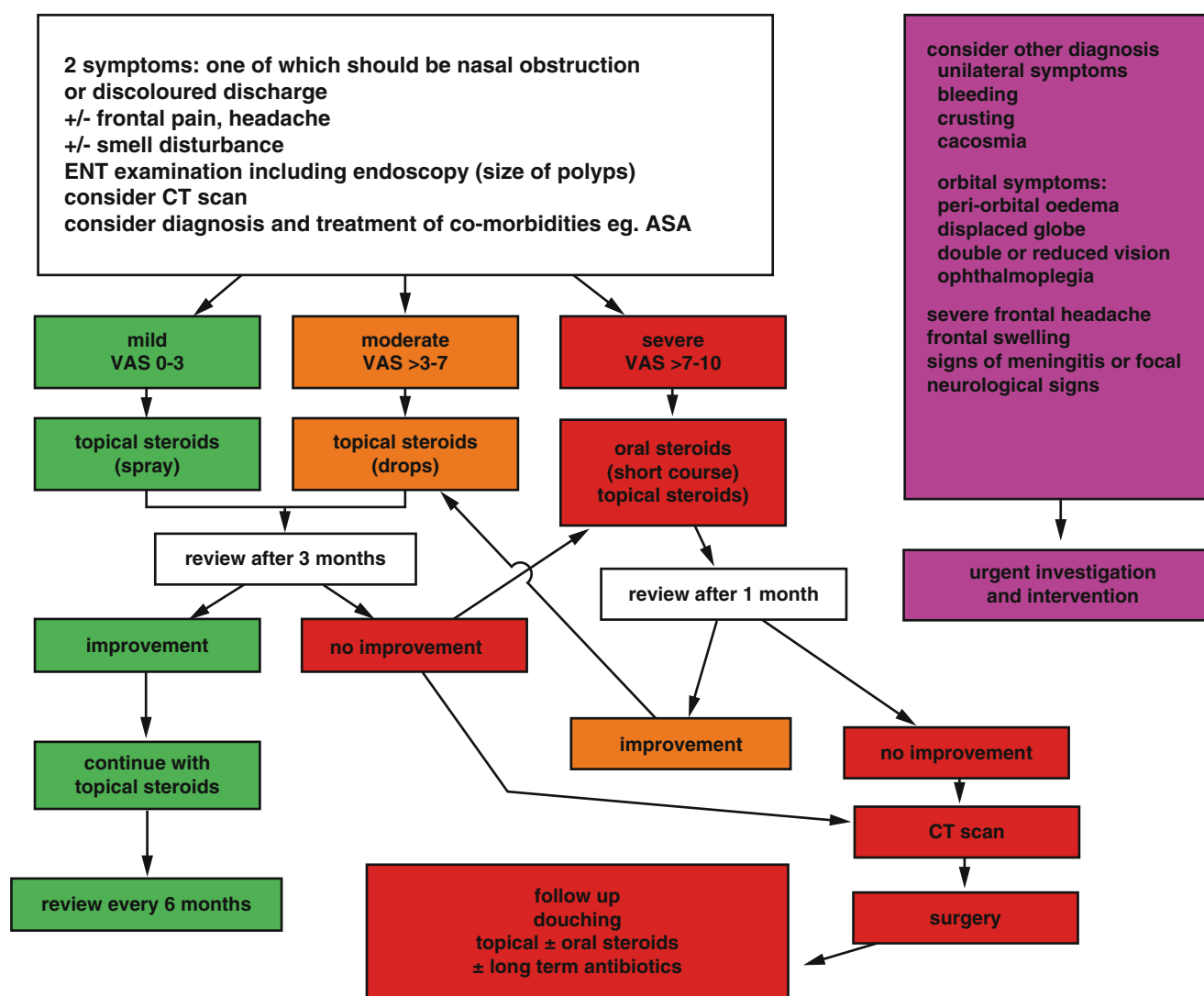


Table 2.7.3 Treatment scheme for ENT specialists for adults with nasal polyps. After Fig. 13.5 of European Position Paper in Rhinosinusitis and Nasal Polyposis, Suppl 20, 2007. Reproduced with permission of *Rhinology*

2.8

Nasal Obstruction

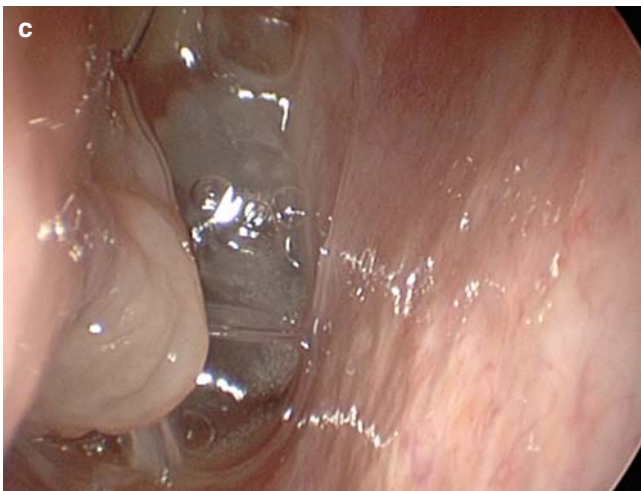
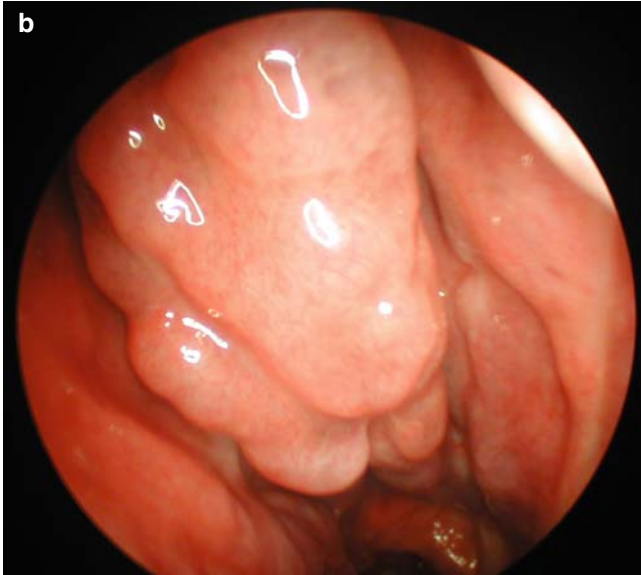
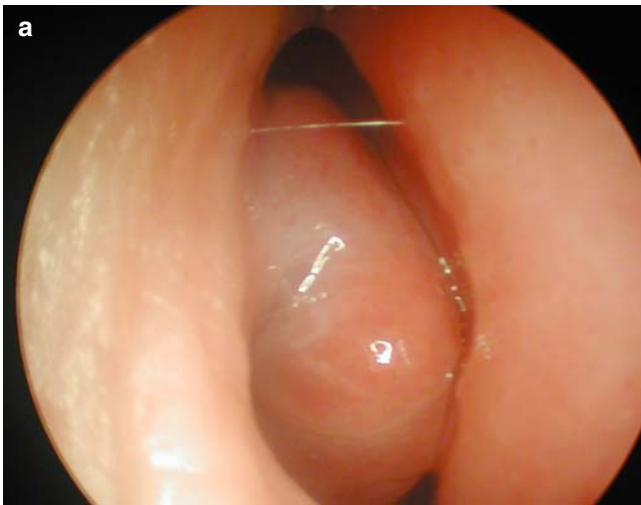


Fig. 2.8.1 (a) Right inferior turbinate hypertrophy. (b) Nodular type left inferior turbinate hypertrophy. (c) Posterior part of the inferior turbinate is polypoid causing nasal obstruction



Fig. 2.8.2 Septal deviation to the right



Fig. 2.8.3 Nasal obstruction due to alar insufficiency, which is the result of overexcision of the lateral crura of the lower lateral cartilages during rhinoplasty. The depressions on both sides lateral to the nasal tip are due to overresection of the alar cartilages



Fig. 2.8.4 Bilateral concha bullosa. No surgery is necessary for asymptomatic cases

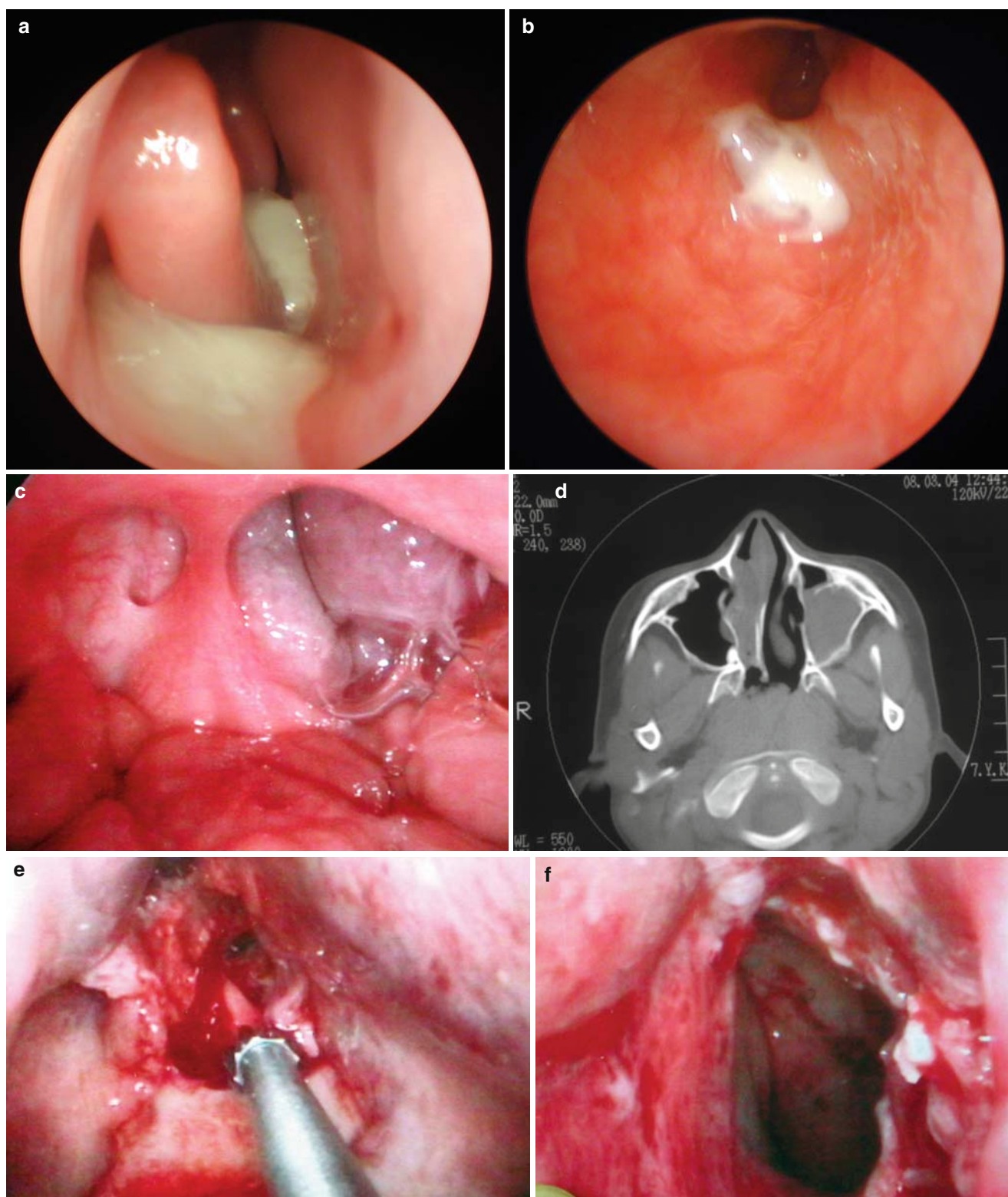


Fig. 2.8.5 Choanal atresia is a congenital abnormality. A bony plate or a membrane obstructs the posterior nares. Unilateral atresia may not cause symptoms. However, bilateral choanal atresia presents an emergency situation since the newborn is totally dependent on the nasal airway for breathing. During feeding, the newborn becomes cyanotic. The diagnosis is made by the inability to pass a soft catheter perinasally, or demonstrating the atretic plate after instillation of radiopaque dye. CT scan shows the atretic plate. The atretic plate can be seen on endoscopic examination. As soon as the diagnosis is made, a transnasal airway should be established. Blindly perforating

the bony plate or the membrane should be avoided because of the narrow nasopharynx and unsatisfactory results. Endoscopic transnasal surgery of choanal atresia gives better results. **(a)** Mucoid discharge in the nasal cavity. **(b)** Endoscopic view of atretic plate from inside the nose. **(c)** Right-sided unilateral choanal atresia; view from the nasopharynx. **(d)** Axial CT scan shows right-sided unilateral choanal atresia. Note the narrow nasal cavity on the atresia side due to thickened pterygoid plates. **(e)** Mucosal flaps have been elevated and the bony plate is drilled. **(f)** Complete opening of the atresia; the flaps are placed in position, the nasopharynx is seen

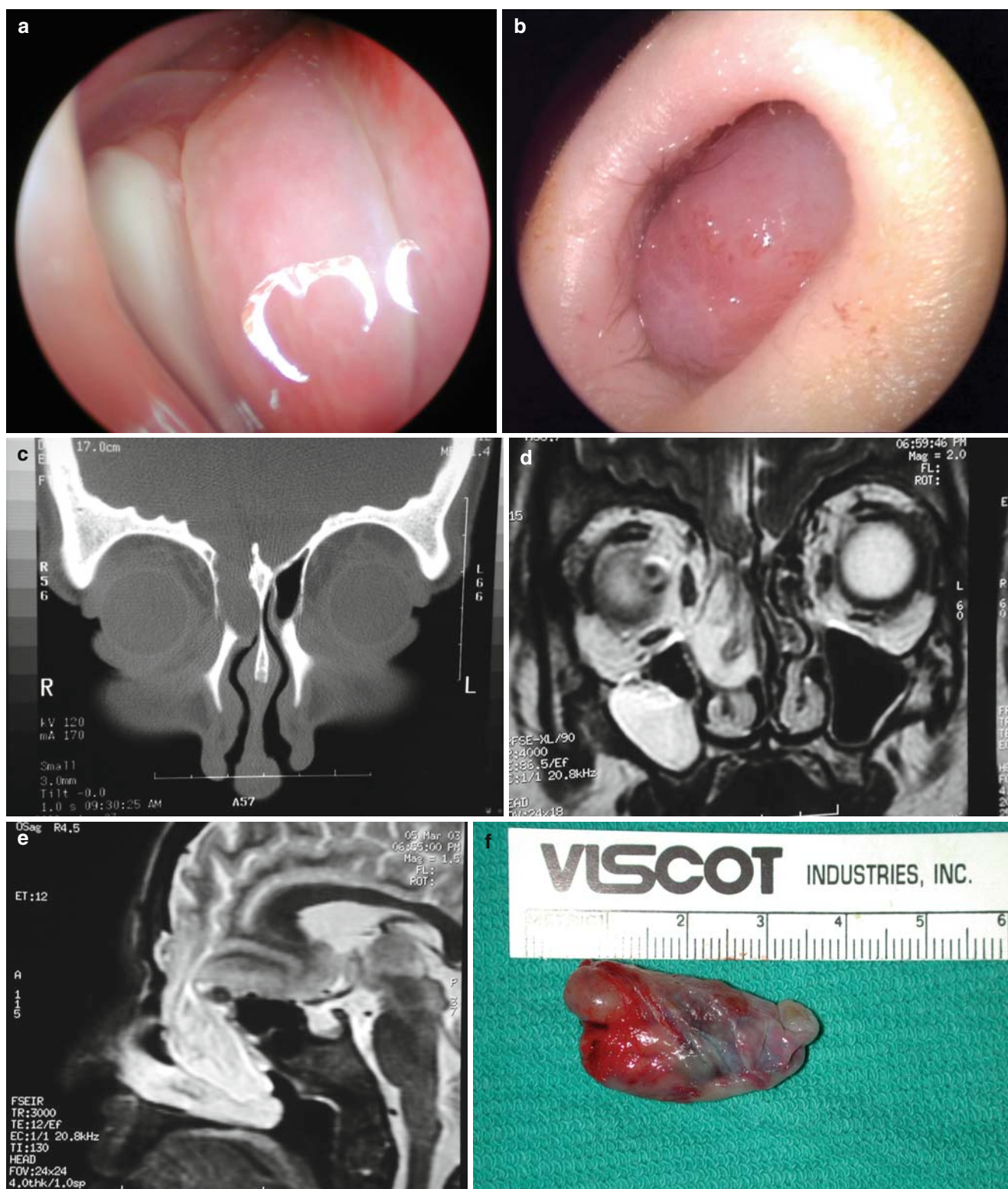


Fig. 2.8.6 Nasal encephaloceles are rare lesions. The brain and meninges herniate through a defect generally in the lamina cribrosa. Encephaloceles are bluish, pulsatile, compressible masses. CT and MR imaging is necessary. Treatment comprises surgical removal of the encephalocele and closure of the defect. **(a, b)** Nasal view of

encephaloceles. **(c)** On the coronal CT scan, the defect at the ethmoid roof is visible. **(d, e)** Coronal and sagittal MR images of the encephalocele herniating through the cranial defect into the nasal cavity. **(f)** Encephalocele after resection. **(g)** Closure of the defect with temporalis fascia

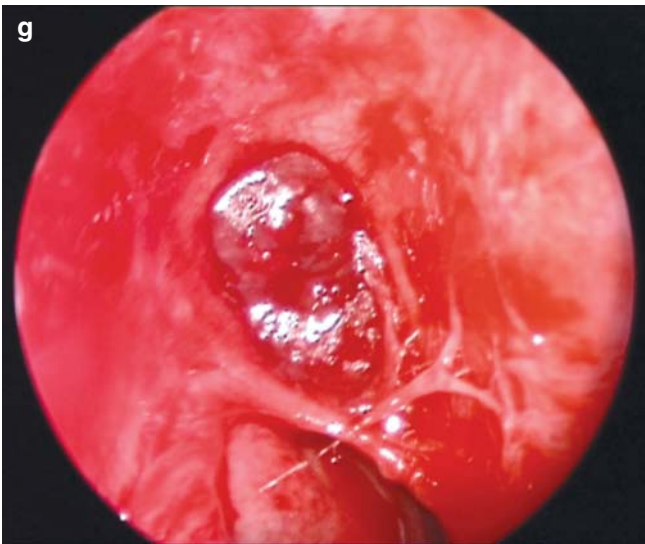


Fig. 2.8.6 (Continued)



Fig. 2.8.7 Empty nose. Although bilateral functional endoscopic sinus surgery and total inferior turbinate resection were performed in a previous operation, the patient felt that his nose was still obstructed

Table 2.8.1 Nasal obstructions

Rhinitis (acute, chronic)
Mechanical factors
Nasopharyngeal diseases (Thornwald cyst, adenoid vegetation)
Turbinate pathologies
Middle turbinate pathologies (paradox middle turbinate, concha bullosa)
Inferior turbinate hypertrophy
Anatomic abnormalities
Septal deviation, septal abscess
Alar collapse
Nasal valve insufficiency
Choanal atresia
Foreign bodies
Nasal masses
Nasal polyps
Encephalocele
Benign tumors
Malignant tumors

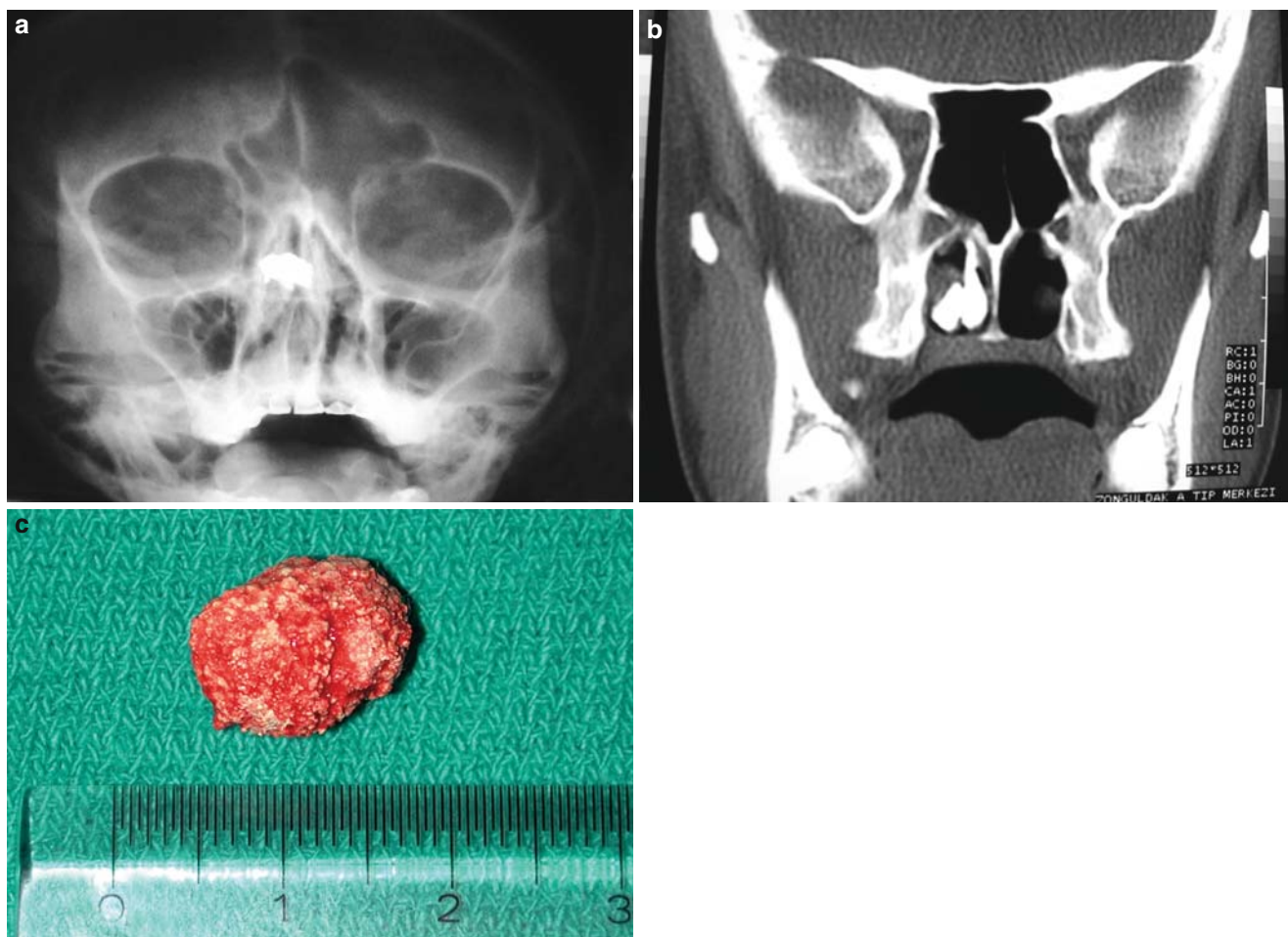


Fig. 2.8.8 Rhinolith. A rhinolith is a large foreign body with deposits of Ca and Mg around a nidus. On examination there is a unilateral mass that is hard on palpation. Radiologic examination helps to make the diagnosis. **(a)** Waters view; an opaque foreign body lateral to the

middle turbinate. **(b)** Coronal paranasal sinus CT showing opaque foreign material inferior and lateral to the inferior turbinate. **(c)** The rhinolith after removal

2.9

Septum

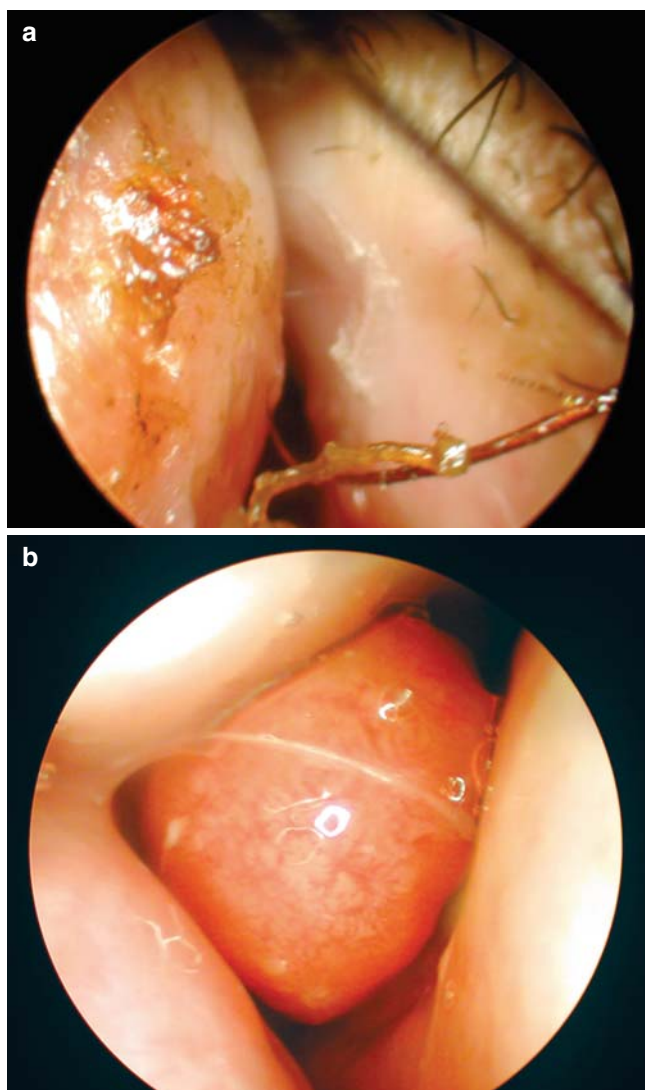


Fig. 2.9.1 (a) Septal deviation to the left side. (b) Compensatory inferior turbinate hypertrophy on the opposite side

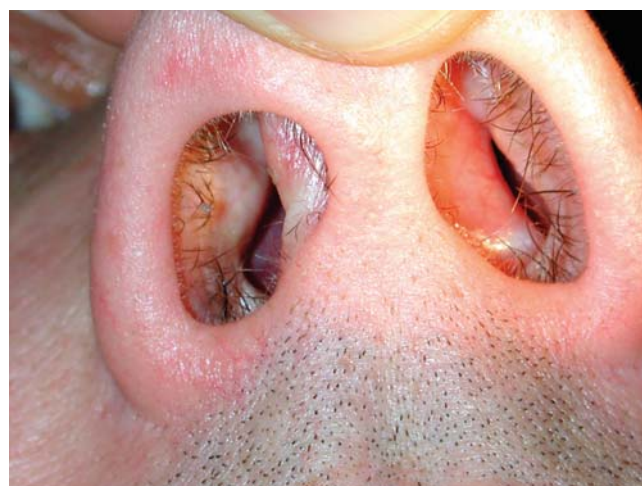


Fig. 2.9.2 Septal spur at the anterior septal area completely obstructing the airway



Fig. 2.9.3 Severe caudal dislocation of the septum to the right

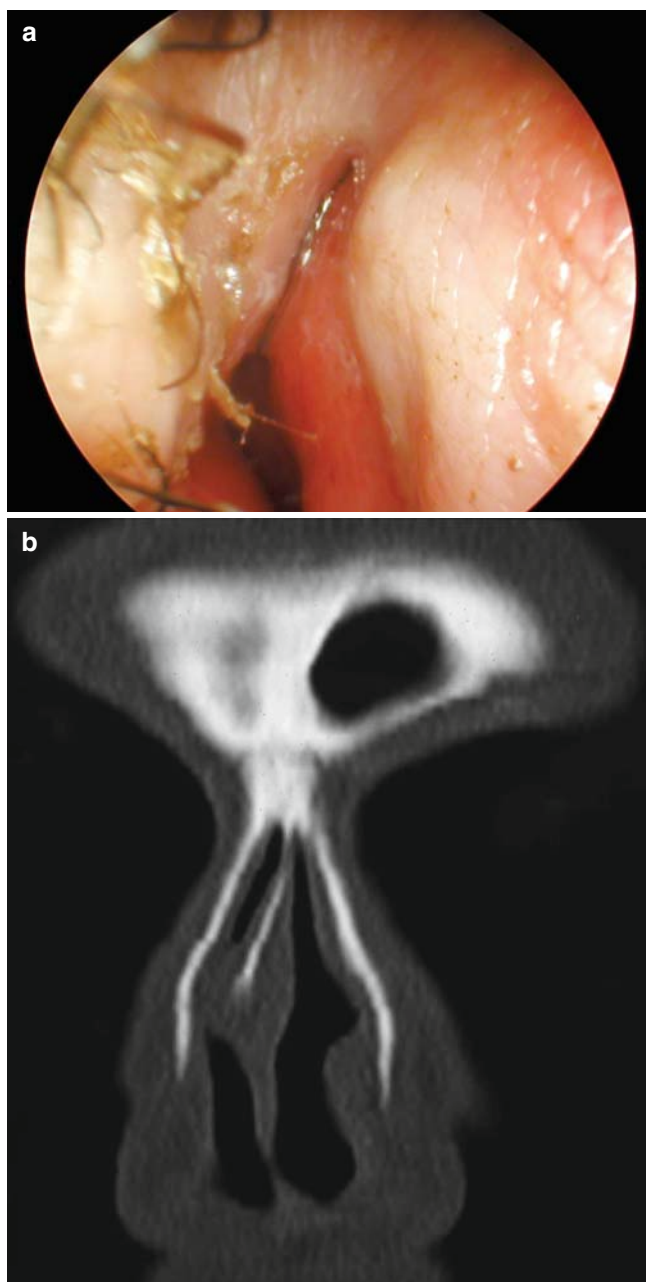


Fig. 2.9.4 (a) Septal deviation to the right, which narrows nasal valve area. (b) Coronal CT shows narrowing of the nasal valve area

Fig. 2.9.6 Septal perforation. There are many causes of septal perforation. The most common cause is previous septal surgery. Other causes include chronic trauma such as nose-picking, use of cocaine, septal hematomas, and infections like tuberculosis. The majority of perforations are located in the anterior cartilaginous portion of the septum. However, syphilitic infection involves the posterior bony septum. In small perforations a whistling sound may be heard during inspiration. Crusting may cause obstruction. As the crusts break off, bleeding occurs. Steam inhalations, nasal douching, and softening ointments may decrease the crusting. (a) Septal perforation after submucous resection. (b) Septal button to close the perforation temporarily. Small and medium-sized perforations can be closed by surgery. For bigger perforations, a silastic nasal septal button can be used for occlusion

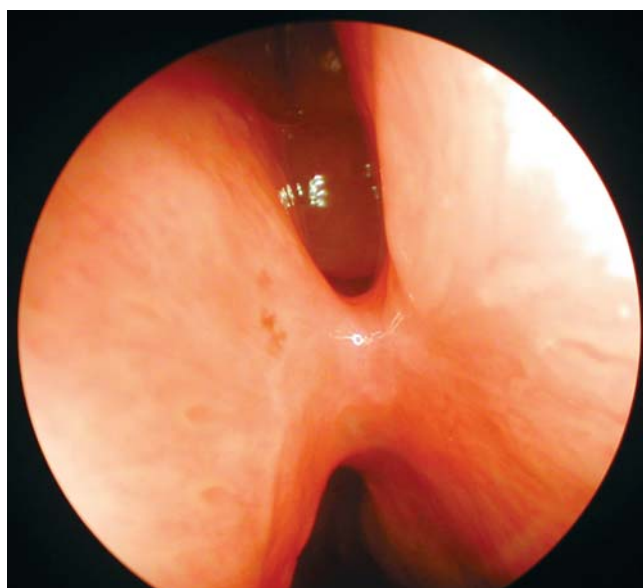


Fig. 2.9.5 Synechia between septum and inferior turbinate

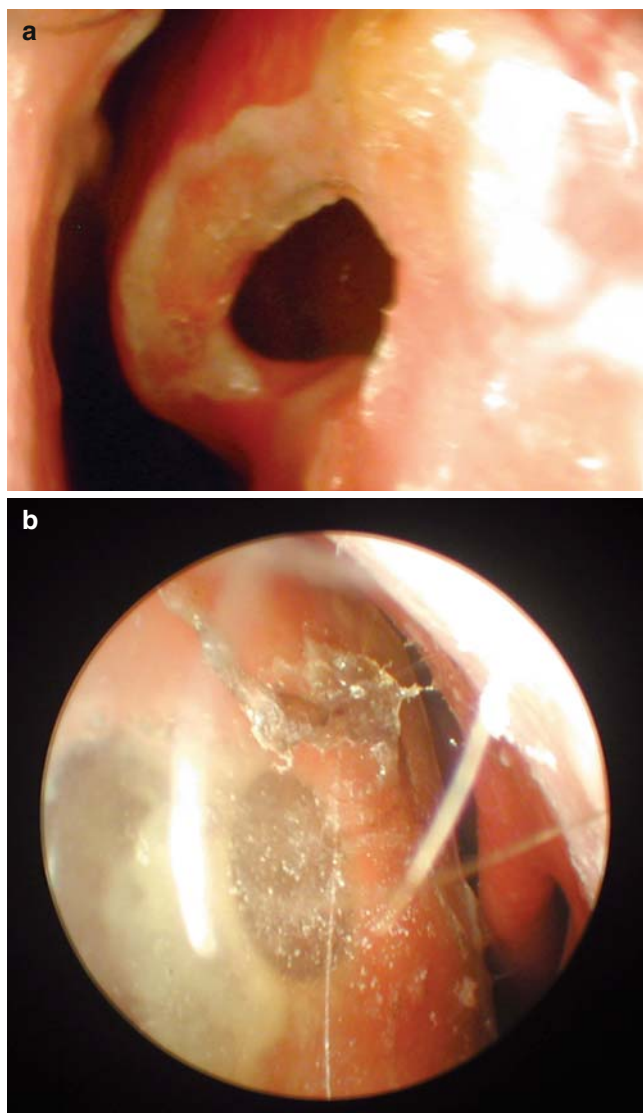




Fig. 2.9.7 (a, b) Saddle nose deformity. This was due to dorsal collapse of the nose as a result of cartilage destruction in a patient with tuberculosis

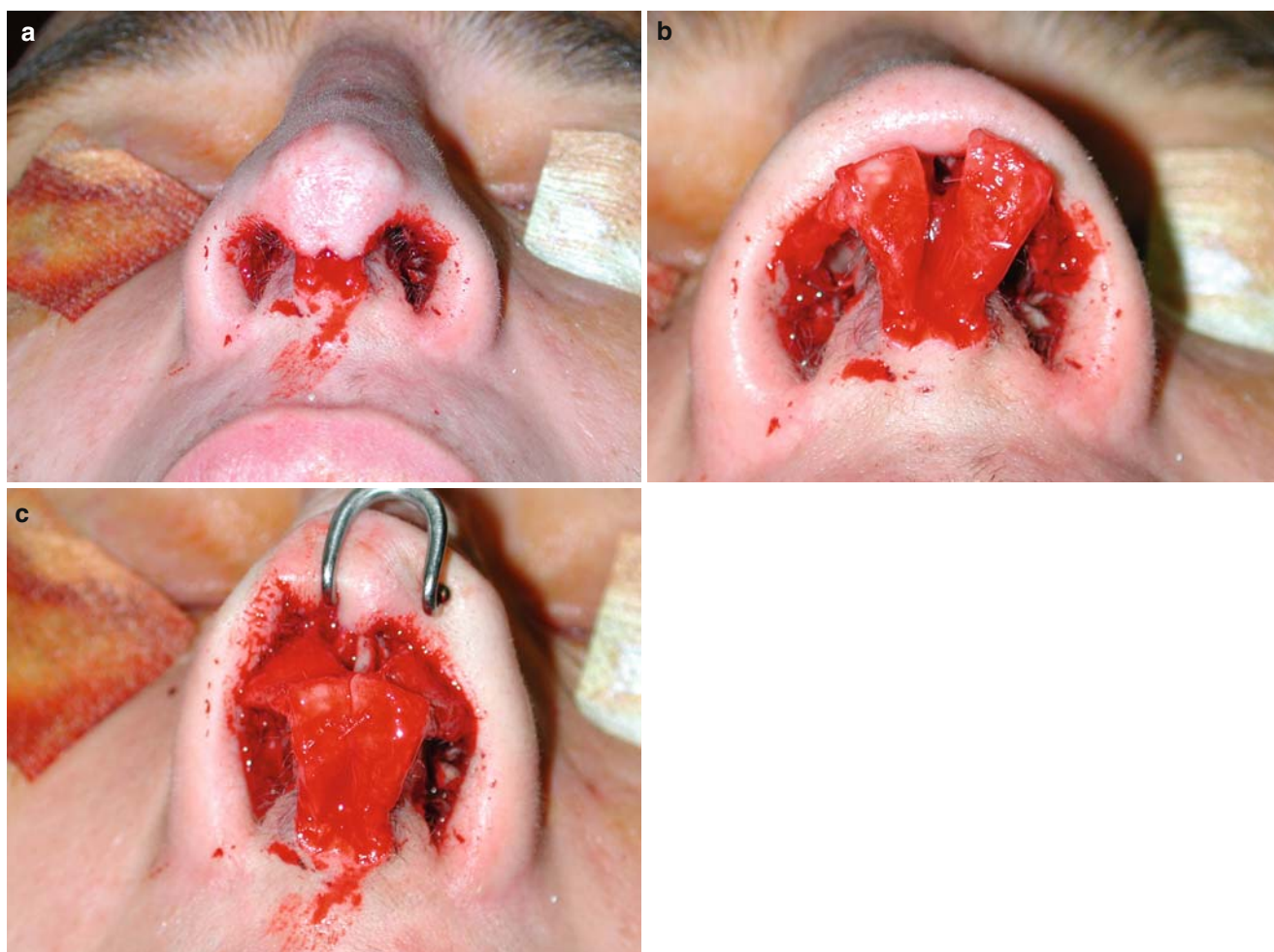


Fig. 2.9.8 External rhinoplasty. (a) A transverse incision across the columella, (b) elevation of the nasal skin superiorly. (c) Suturing lower lateral cartilages

Fig. 2.9.9 (a) Cartilage and bony hump removal. (b) To rotate the nasal tip superiorly, the septal cartilage, lower and upper lateral cartilages may be shortened. (c) Medial and lateral osteotomies to narrow the nose

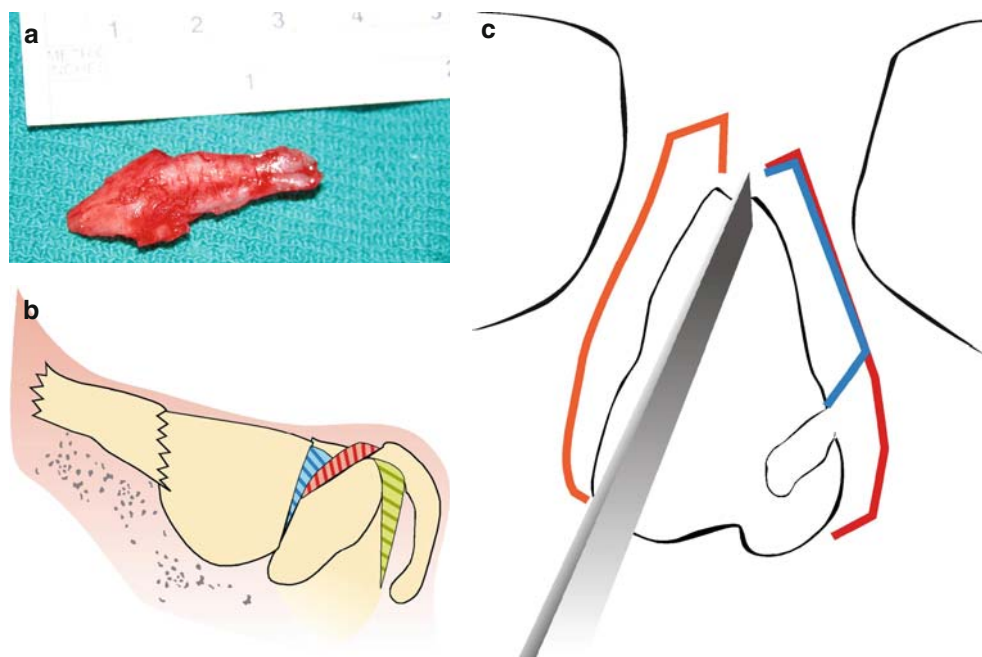


Fig. 2.9.10 Infected cartilage graft at the nasal dorsum after rhinoplasty

2.10

Epistaxis

Although the majority of cases of epistaxis are self-limiting, it may sometimes be very severe and very difficult to manage. In Table 2.10.1, local and systemic factors that may cause epistaxis are presented.

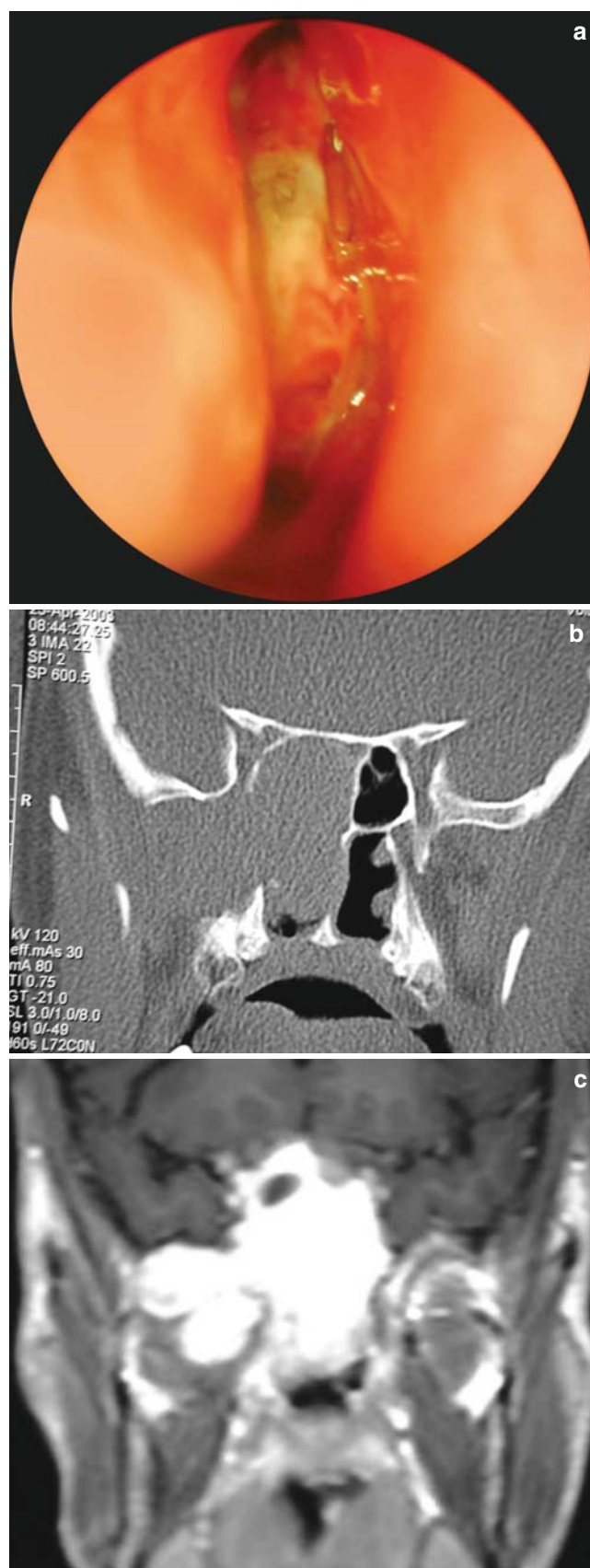
Epistaxis may be from the anterior or posterior septum or nasal cavity. Posterior epistaxis is usually from the sphenopalatine artery and sometimes may be difficult to control and manage. Bleeding from Little's area may be cauterized by silver nitrate.

The patient sits in the upright position. The head should be kept straight and should not be extended in order to keep the intracranial pressure low. The nose should be cleared of clots by blowing. Cotton wool pledgets soaked in appropriate solution are placed in the nasal cavity. In hypertensive patients, adrenaline should not be used. These pledgets show whether the bleeding is from the anterior or posterior side. If the bleeding is from Little's area, it can be cauterized. Anterior packing should be placed in layers. If the epistaxis is from the posterior septal area, then posterior packing may be necessary. Systemic diseases should be treated.

Table 2.10.1 Etiology of epistaxis

Local
Trauma
Inflammation
Postoperative
Foreign body
Nasal and paranasal sinus tumors
Hereditary hemorrhagic telangiectasia
Atrophic rhinitis
Systemic
Hypertension
High venous pressure (mitral stenosis)
Blood dyscrasias (leukemia, hemophilia, vitamin K deficiency)
Anticoagulant drugs

Fig. 2.10.1 Unilateral bleeding and nasal obstruction in a young male patient should raise the suspicion of juvenile nasopharyngeal angiofibroma (JNA). (a) Endoscopic view of the JNA in the nose. (b) Coronal CT shows widening of the pterygomaxillary fissure. (c) Hypervascularity of the tumor



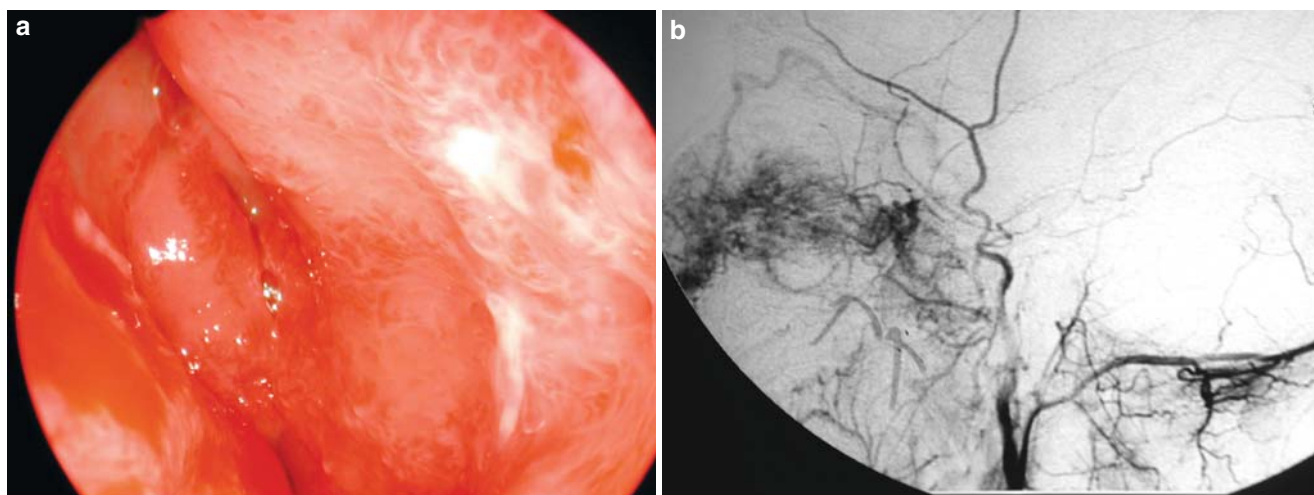


Fig. 2.10.2 (a) Hereditary hemorrhagic telangiectasia (Osler-Weber-Rendu disease) is characterized by small abnormal capillaries in the nasal mucosa. It may cause serious nasal bleeding, and hemoglobin levels may drop to very low levels, necessitating blood transfusion. In

the nasal mucosa, numerous leashes of bleeding vessels can be seen. Laser coagulation may be effective in the early stages. In severe cases, skin grafting may be necessary. **(b)** Angiography shows hypervascularization

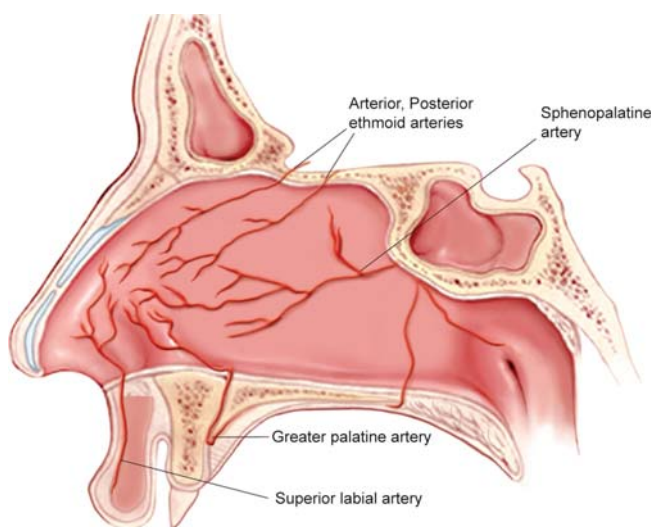


Fig. 2.10.3 Kiesselbach's plexus (Little's area). It is localized at the anterior portion of the septum. Approximately 90% of bleeding occurs from this area. The external and carotid artery systems anastomose in this area. The anterior and posterior ethmoid arteries from the internal carotid artery system and the superior labial artery, greater palatine artery, and sphenopalatine artery from the external carotid artery system form a vascular plexus



Fig. 2.10.4 Bleeding vessels in Kiesselbach's plexus (Little's area)



Fig. 2.10.5 Cauterization of Little's area with silver nitrate sticks

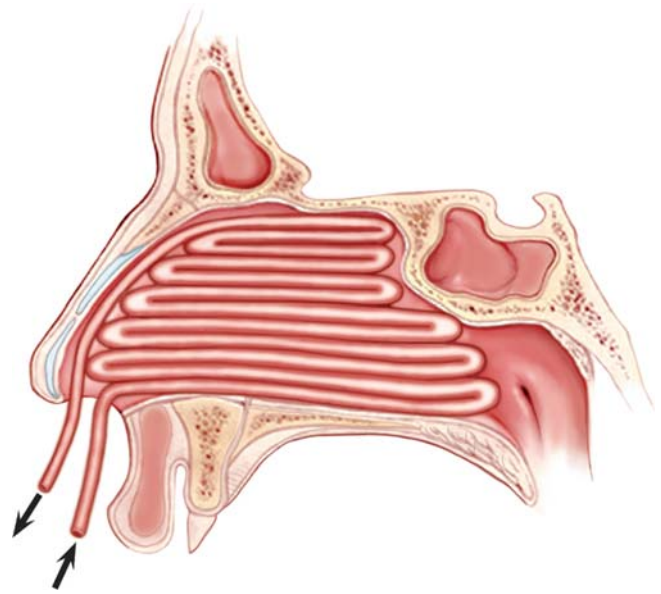
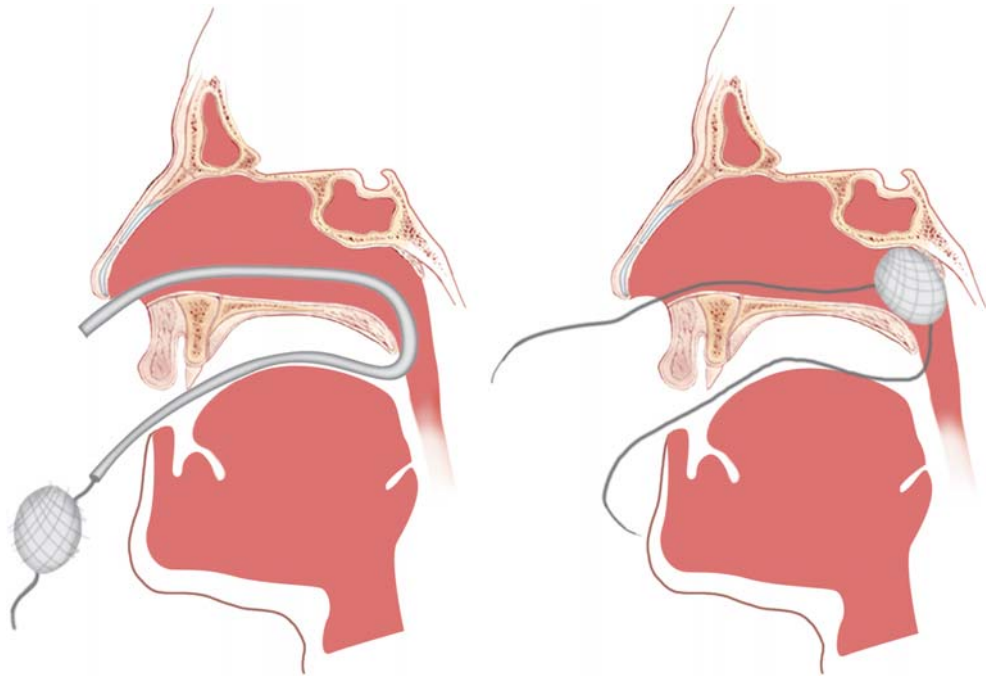


Fig. 2.10.6 Anterior nasal packing with Vaseline-impregnated gauze. After cleaning the clots, the packing is done in layers

Fig. 2.10.7 Posterior nasal packing. A postnasal pack is prepared from a tightly compressed ball of gauze according to the size of the postnasal space and tied with 0-silk ties. A soft rubber catheter is passed along the floor of the nose to the pharynx and taken out through the mouth. One end of the silk tie is attached to the tip of the catheter and drawn back through the nose. The pack is pushed behind the soft palate in the nasopharynx by exerting moderate pressure. The ends of the silk ties are attached near the nose and mouth corner. All patients with posterior nasal packing should be hospitalized for close observation



2.11

Traumas



Fig. 2.11.1 Nasal fracture. A blow coming from the lateral side caused the nose to be displaced to the other side. There is ecchymosis in the infraorbital area



Fig. 2.11.2 Lateral X-ray. Nasal fracture shows displacement of nasal bones

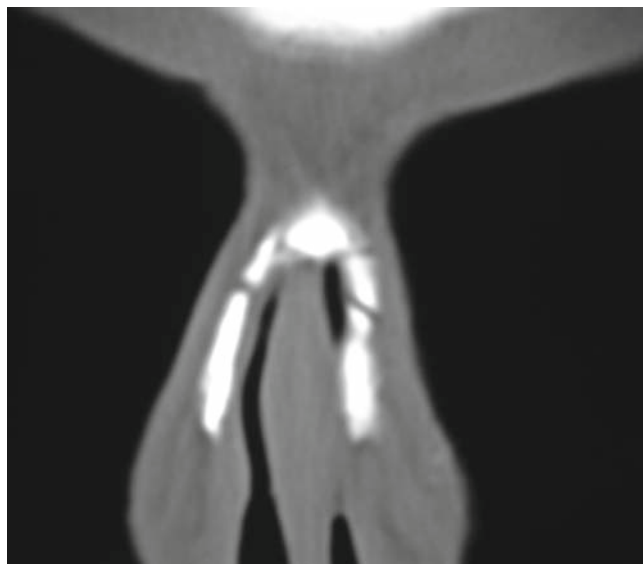


Fig. 2.11.3 Coronal CT scan showing multiple fractures of the nasal bones

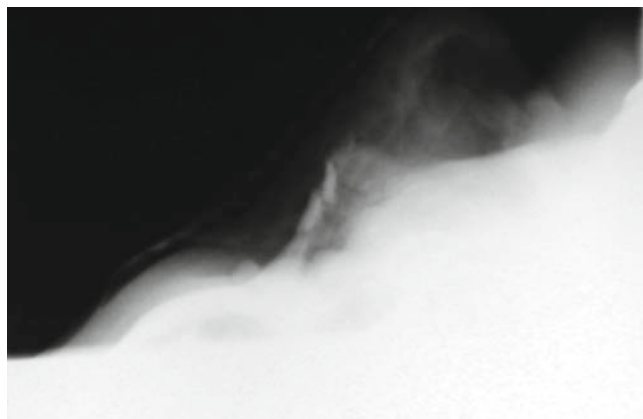


Fig. 2.11.4 Lateral X-ray showing nasal bone fracture. Depression of the nasal bones requires the elevation of the depressed bony fragments



Fig. 2.11.5 Raccoon eyes or periorbital ecchymosis is a sign of basal skull fracture. It results from blood from the skull fracture tracking down into the soft tissue around the eyes (courtesy of Dr. Kiratli)

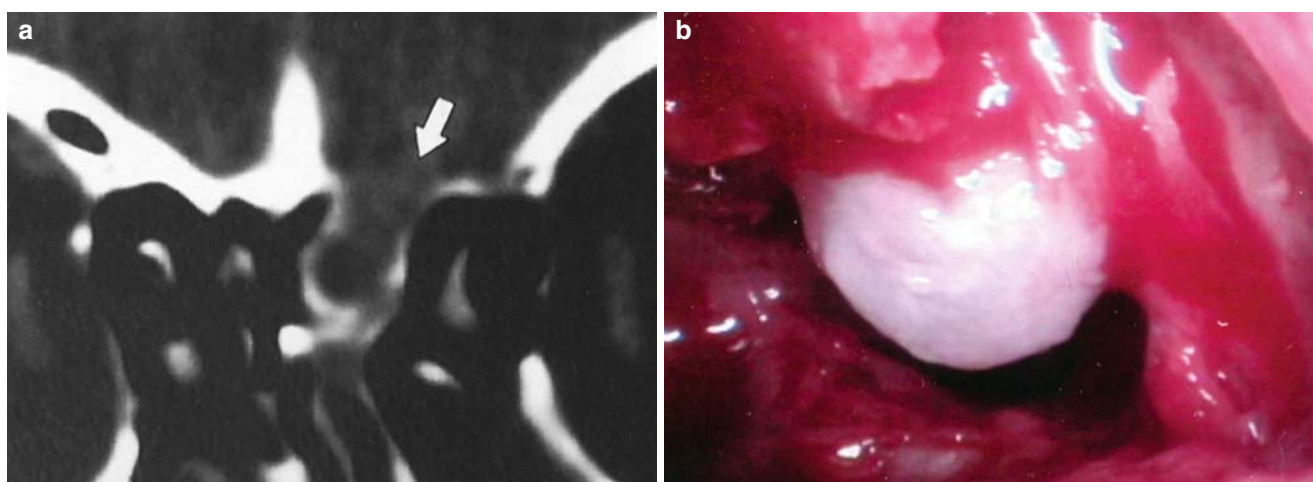


Fig. 2.11.6 Cerebrospinal fluid rhinorrhea after trauma. **(a)** Coronal CT showing herniation through the lamina cribrosa at the anterior ethmoid area. **(b)** Photograph of the herniated tissue

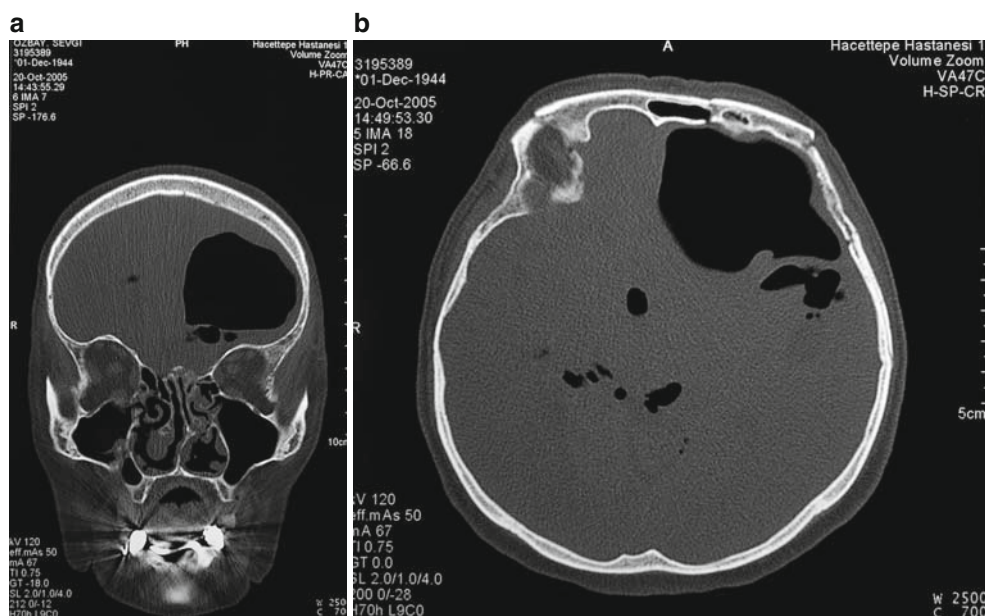


Fig. 2.11.7 (a, b) Air in the intracranial cavity after traumatic fracture in the anterior and posterior frontal tables and anterior skull base



Fig. 2.11.8 (a, b) Le Fort III maxillary fracture. Extensive facial fractures may require urgent intervention to prevent respiratory obstruction. Maxillary fractures were first classified by Dr. Le Fort and this classification system was called the “Le Fort classification.” In the Le Fort classification system there are three types of fractures: Le Fort I: The fracture line passes along the pyriform aperture and inferior wall of the antrum. It is a low maxillary fracture that separates the maxilla at the level of

the nasal floor. Le Fort II (pyramidal fracture): The fracture line passes across the nasal bone, the frontal processes of the maxilla, and the zygomatic-maxillary junction. It separates the *central (middle)* third of face from the base of the skull. Le Fort III (craniofacial dysjunction): The facial bones separate completely from the skull base. The fracture line extends along the nasofrontal, maxillofrontal, and zygomaticofrontal sutures. These fractures are reduced with miniplates

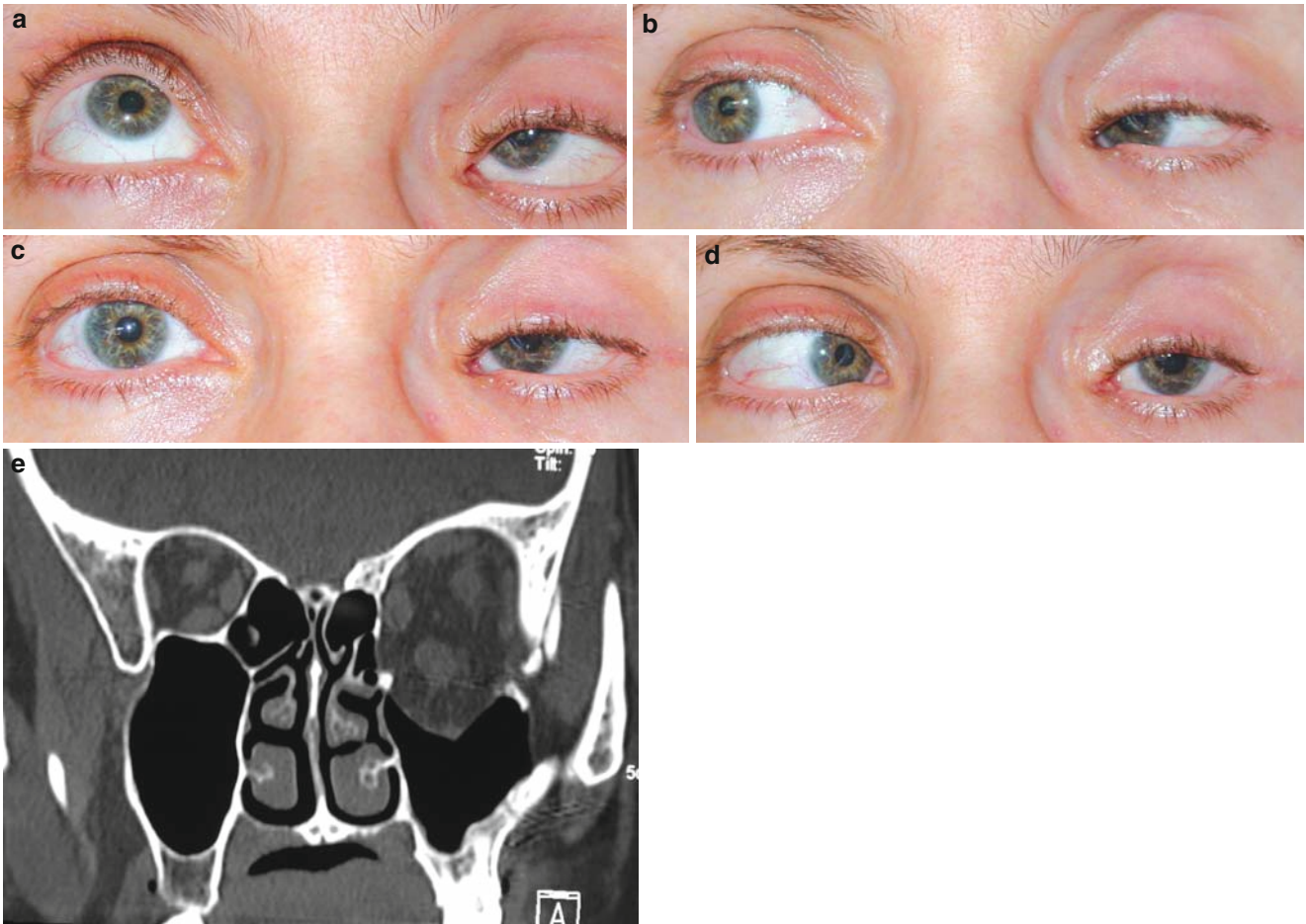


Fig. 2.11.9 Left-sided orbital blowout fracture. The soft tissue contents of the orbit have extruded into the maxillary antrum. Orbital blowout fracture is usually caused by blunt trauma to the eye globe. Increased pressure in the orbit causes fracture in the weakest part of the orbital cavity. The orbital contents extrude into the maxillary antrum. Entrapment of the inferior rectus muscle causes limitation of

eye movement. Orbital herniation leads to enophthalmos on long-term follow-up. (a–g) After a severe maxillofacial trauma, mistreated blowout fracture resulted in enophthalmos and restriction in the mobility of the left eye. The patient recovered from diplopia after surgery (courtesy of Dr. Şener)

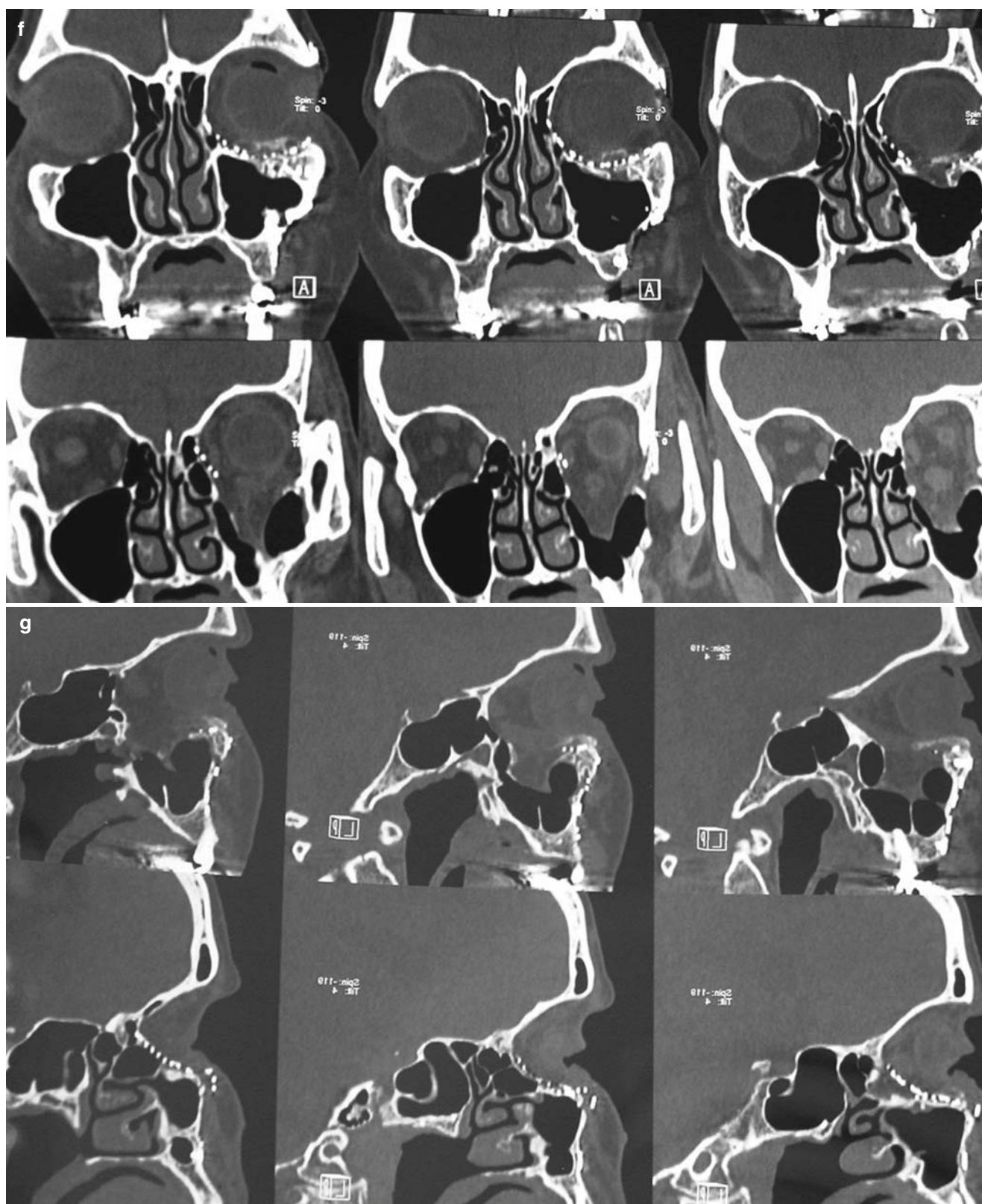


Fig. 2.11.9 (Continued)

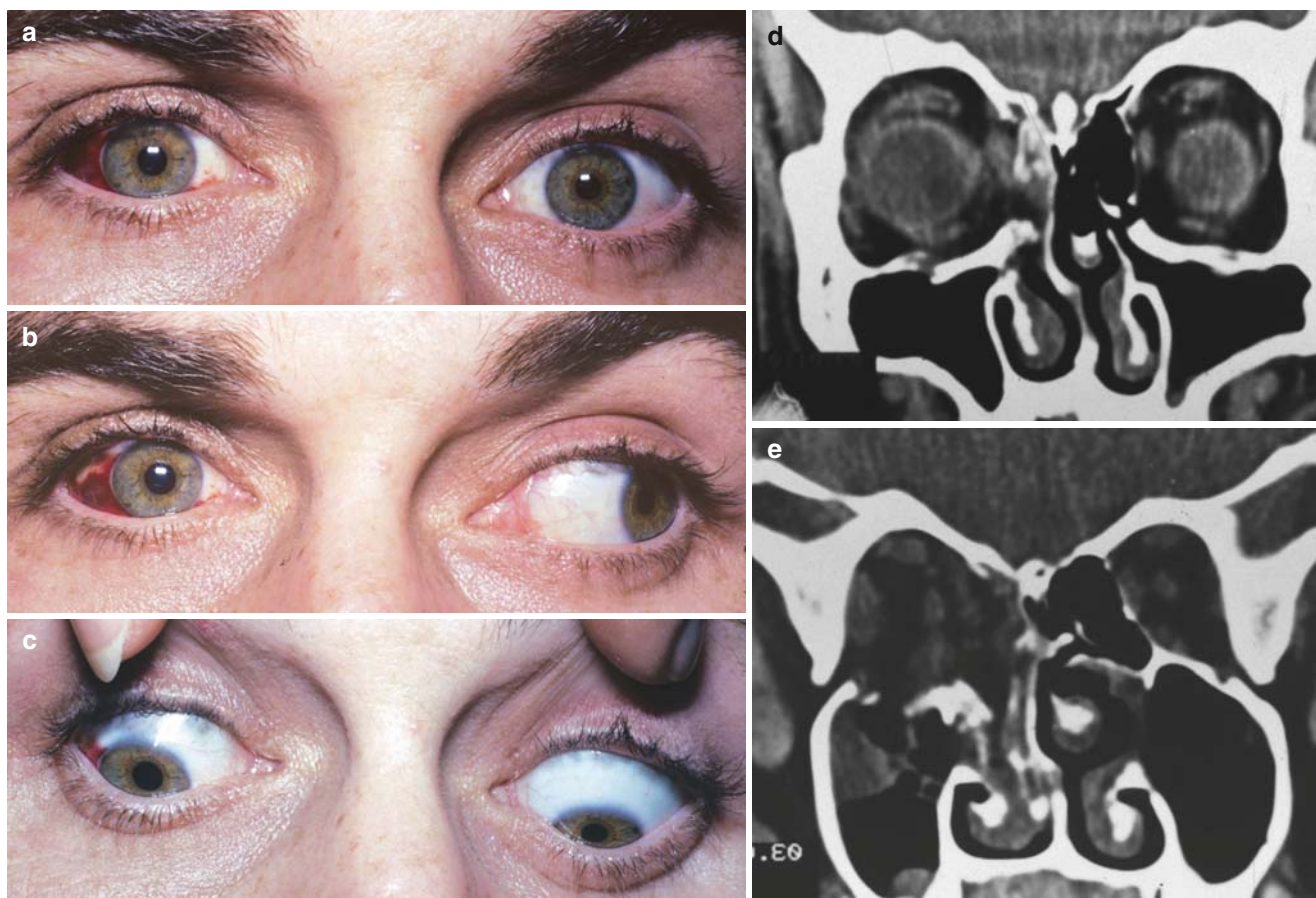


Fig. 2.11.10 (a–e) After a severe traffic accident, fracture in the medial and inferior orbital walls resulted in enophthalmos and restriction in the mobility of the right eye. Gaze to the medial and inferior sides is limited. Note the herniation of the globe to the ethmoids (courtesy of Dr. Şener)

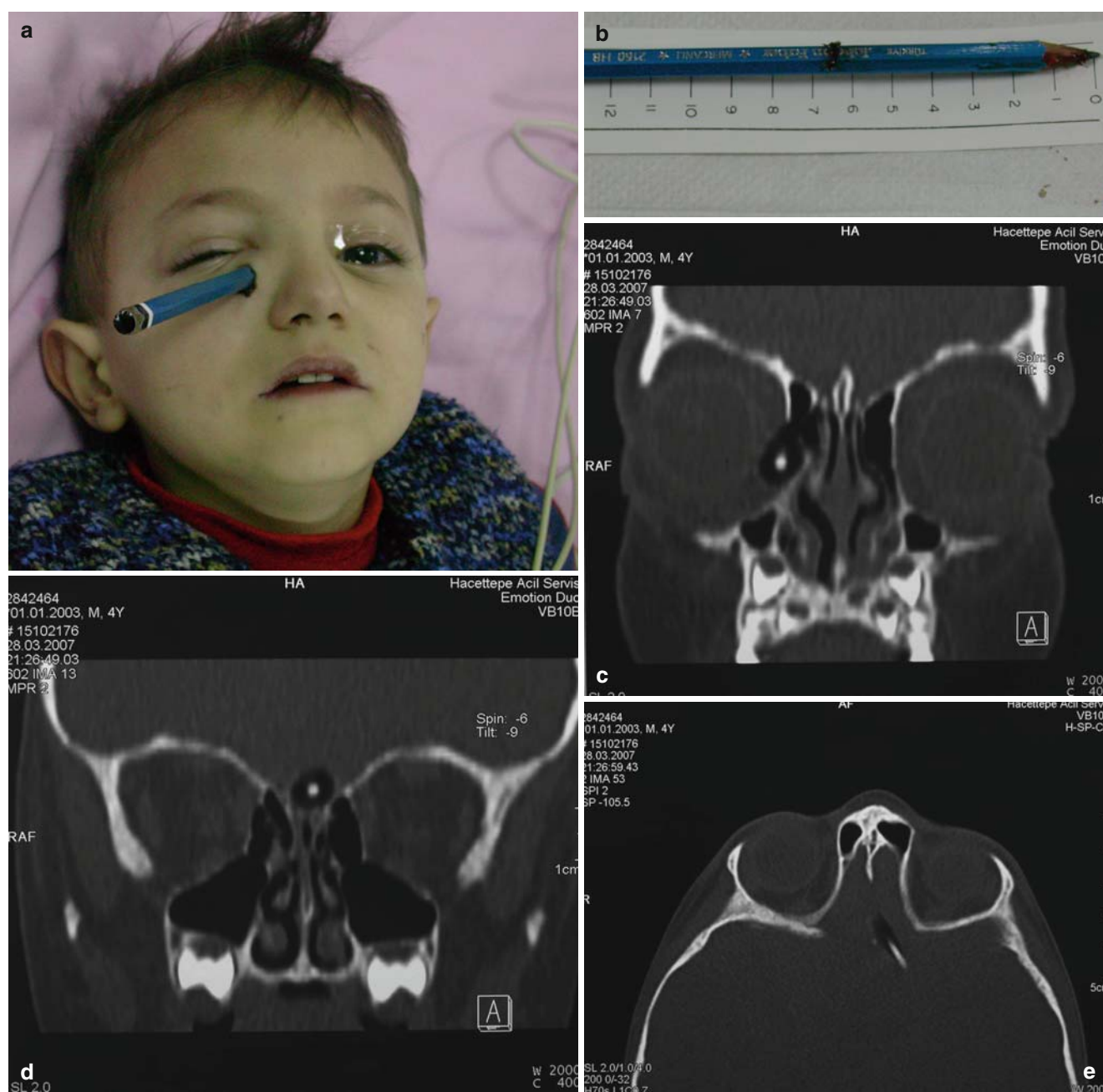


Fig. 2.11.11 Penetrating trauma to the right orbit and the intracranial cavity. The pencil was taken out, all foreign material cleaned, and CSF fistula repaired. **(a, b)** Pencil penetrating the skin just inferior and

medial to the orbit. **(c-e)** Coronal and axial CT scans showing trauma to the eye and the intracranial cavity (courtesy of Tesav)

2.12

Nasolacrimal obstructions



Fig. 2.12.1 Acute dacryocystitis on the left side



Fig. 2.12.2 Acute dacryocystitis with severe infection around the eye. Note the fluctuating mass in the left internal canthal area with hyperemia and edema around the left eye



Fig. 2.12.3 Congenital dacryostenosis with lengthened fluorescein drainage time on the right side

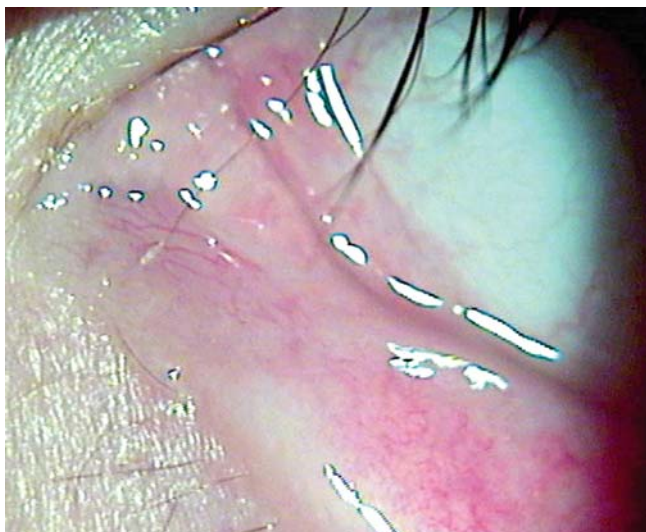


Fig. 2.12.4 Punctum agenesis on the left side



Fig. 2.12.5 Congenital dacryostenosis on the left side. (a) Macro-dacryocystography. Opaque material on the left side was not drained to the nose. Opaque material on the right side has already been drained. (b) Probing of the left nasolacrimal canal. (c) Fluorescein suctioned from the inferior meatus after injection to the nasolacrimal canal

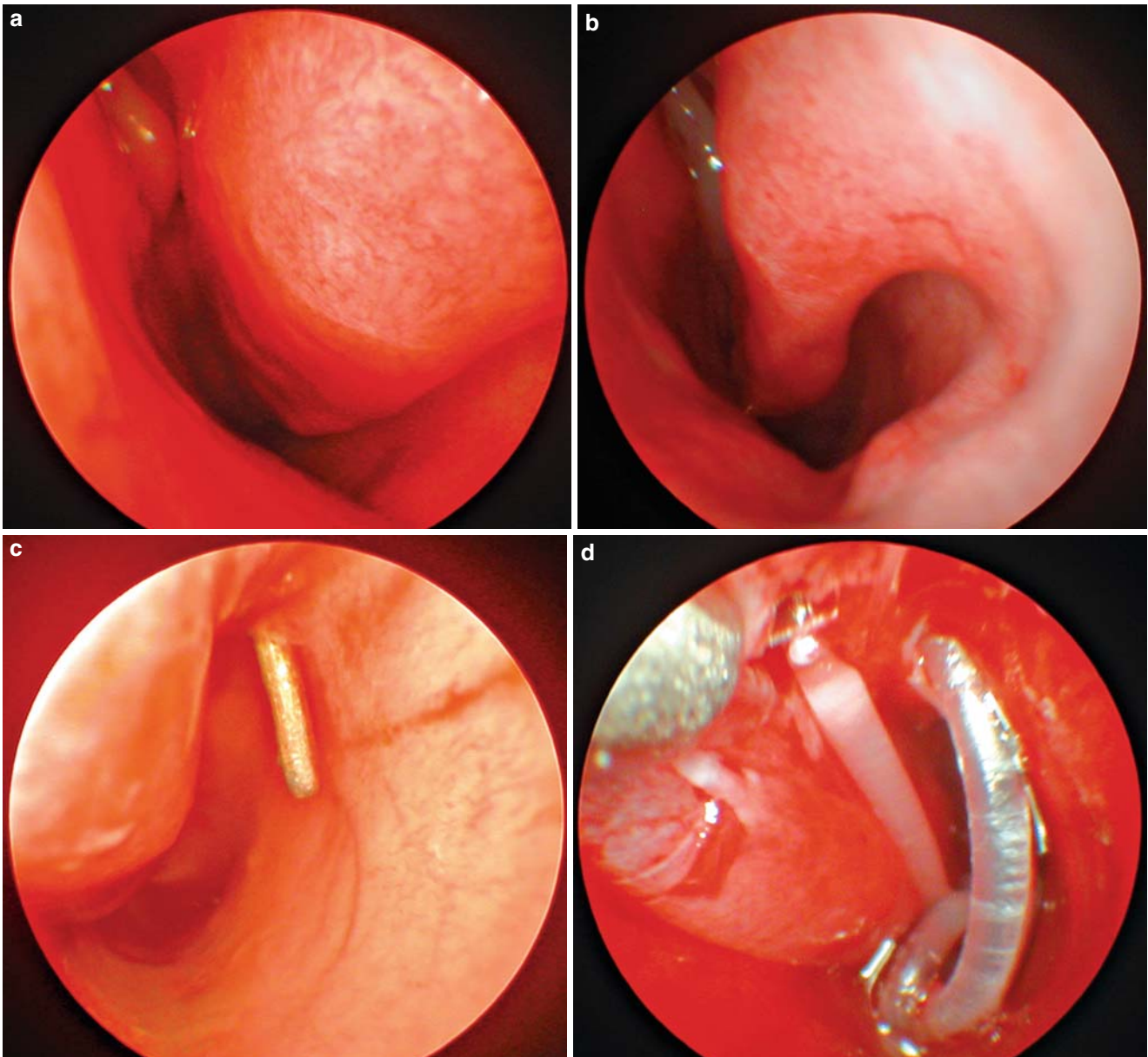


Fig. 2.12.6 Endoscopic intubation of a patient with congenital dacryostenosis on the left side. **(a)** Inferior turbinate. **(b)** Medialization of the inferior turbinate. **(c)** The tip of the probe in the inferior meatus.

(d) Silicon tubes tied in the inferior meatus after they have been passed through the nasolacrimal canal under endoscopic control

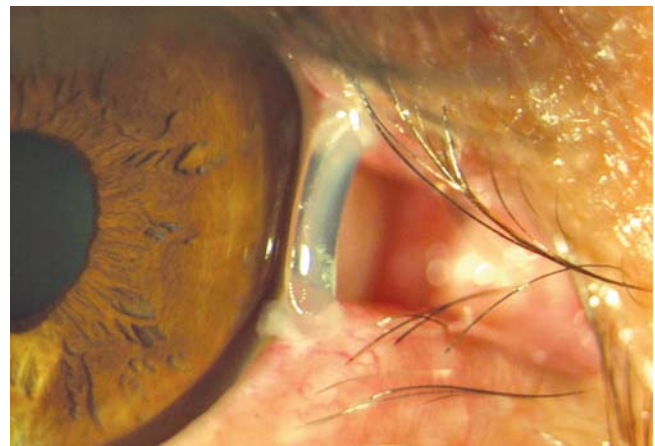


Fig. 2.12.7 The loop of the silicon tube should not be very tight in order to avoid trauma to the canaliculi

2.13

Tumors

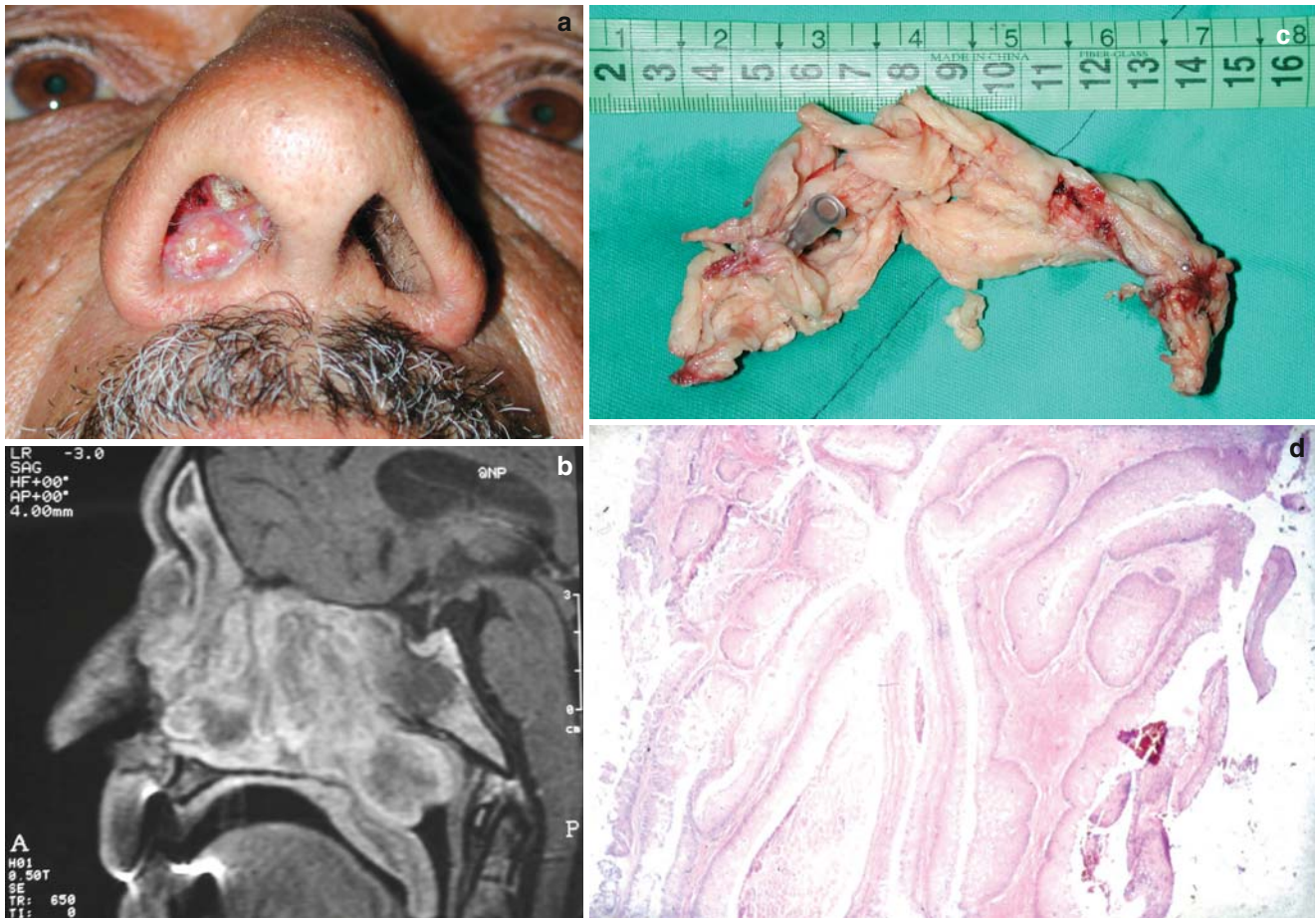


Fig. 2.13.1 Inverted papillomas generally arise from the lateral nasal wall. They are unilateral masses. In 15% of cases there may be malignant transformation. The tumor should be excised completely and sent for histological examination to check for malignant transforma-

tion. Endoscopic removal is the treatment of choice. **(a)** Right-sided mass. **(b)** On sagittal MR image, the mass completely fills the nasal cavity. **(c)** Removal of the mass. **(d)** Histological picture of the inverted papilloma. Epithelium shows invaginations into the tissue

Fig. 2.13.2 Inverted papilloma that fills the nostril and is clearly visible

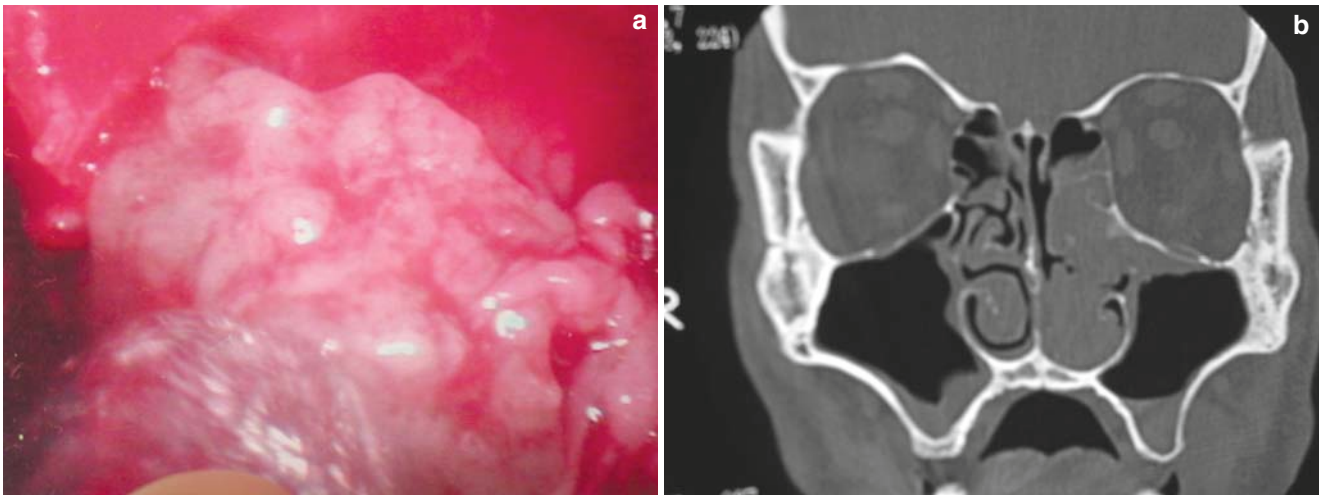


Fig. 2.13.3 Left-sided inverted papilloma. (a) Endoscopic appearance. (b) Coronal CT shows extension to the upper wall of the maxillary antrum

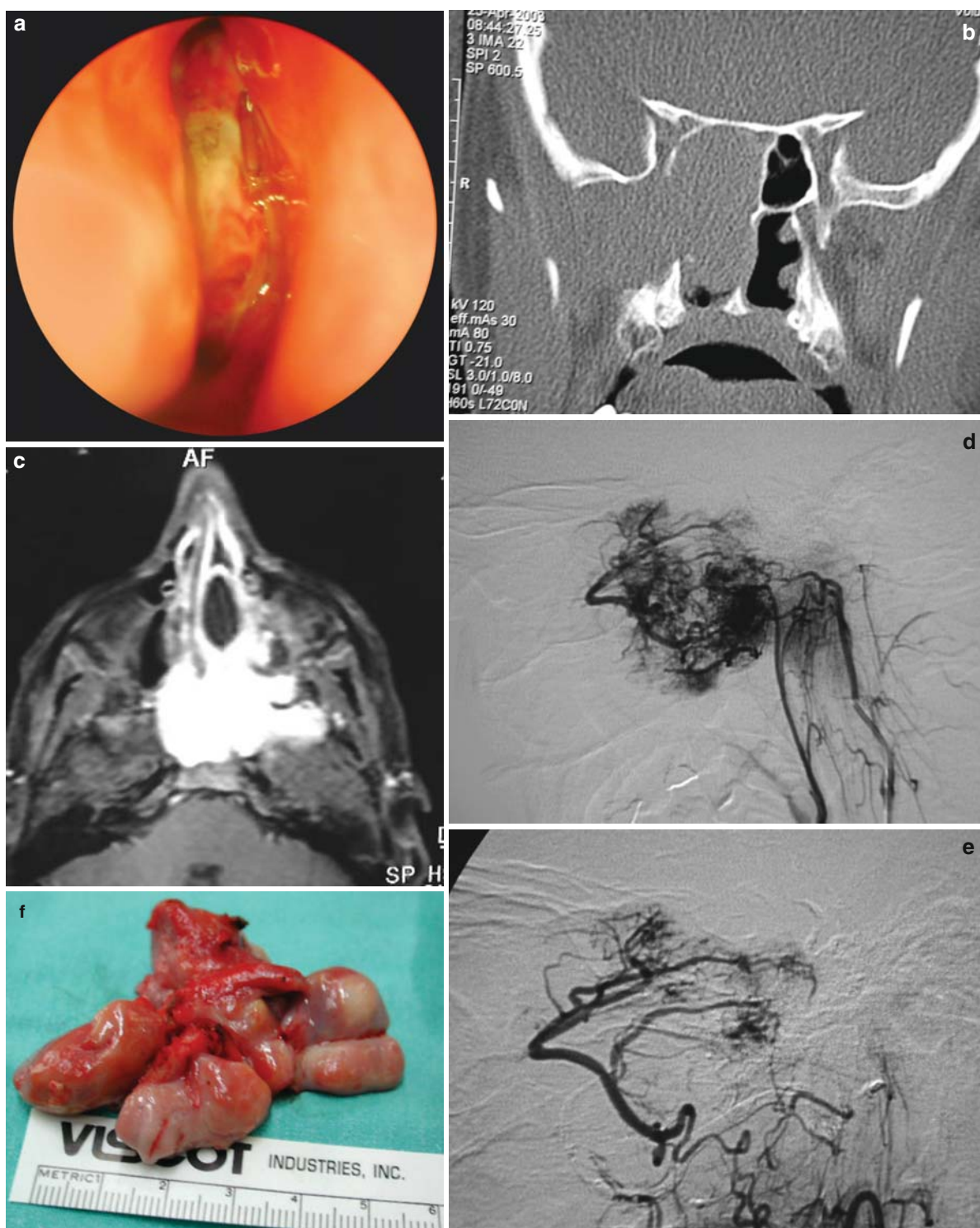


Fig. 2.13.4 Juvenile nasopharyngeal angiofibroma. It usually arises superior and posterior to the sphenopalatine foramen. It only occurs in male adolescents. These patients present with the complaint of nasal obstruction and bleeding. **(a)** Endoscopic view. **(b)** On coronal CT scan, widening of pterygomaxillary fissure is diagnostic. **(c)** Axial CT

shows extension to the infratemporal fossa. **(d)** Angiography shows the hypervascularity of the tumor. **(e)** Embolization helps the surgeon to operate in a less bloody field. No vascularity after embolization is seen. **(f)** Endoscopic surgery is the treatment of choice. Specimen after total removal of the tumor

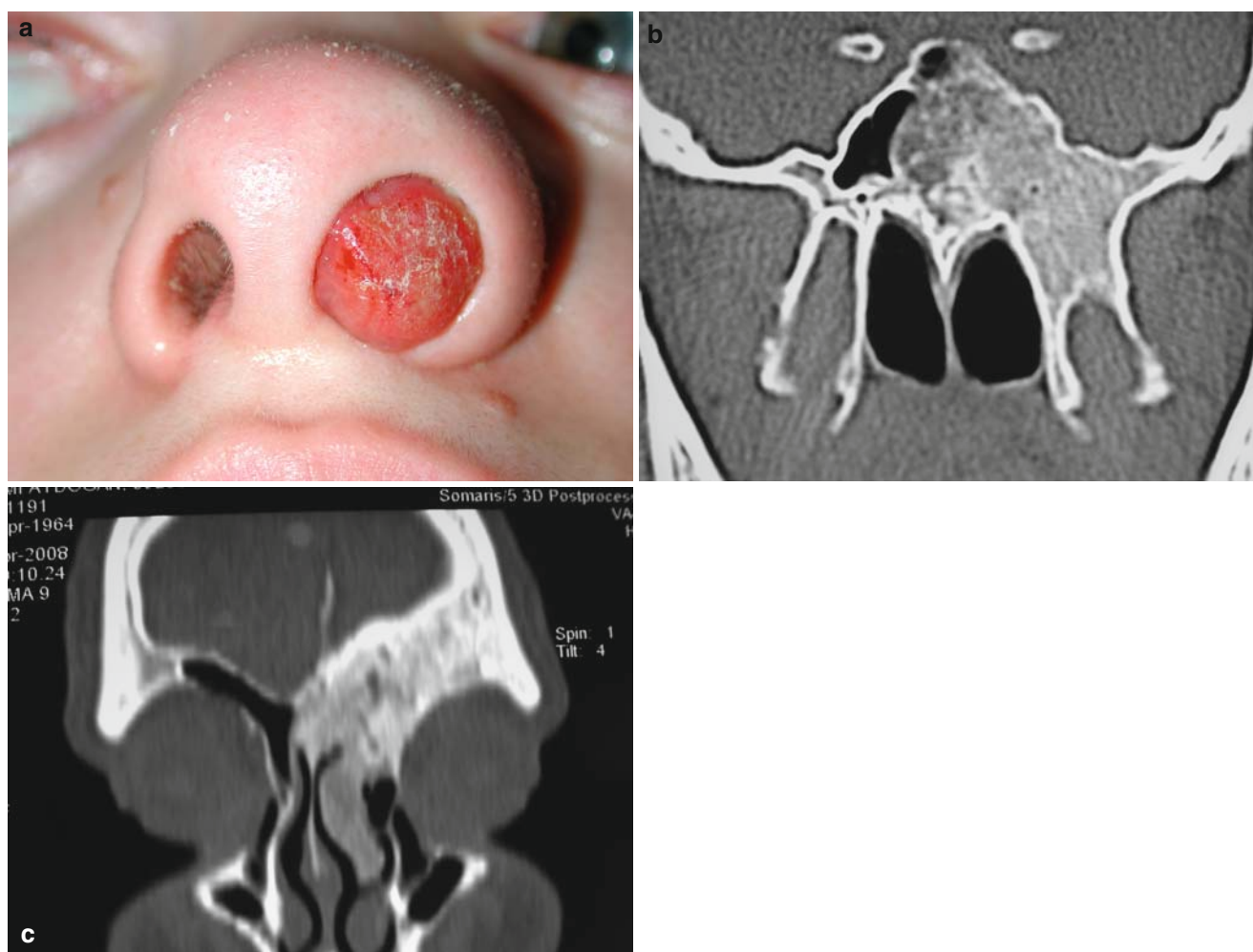


Fig. 2.13.5 Fibrous dysplasia is a fibro-osseous disease of the facial skeleton. It usually appears during the first two decades of life. On CT scan, ground glass presentation due to a mixture of fibrous and osseous components is characteristic of this tumor. If it does not

cause any deformity or functional disorder, no surgery is required. **(a)** Fibrous dysplasia filling the left nostril completely. **(b)** Coronal CT showing fibrous dysplasia in the sphenoid bone. **(c)** Coronal CT showing fibrous dysplasia in the frontal bone

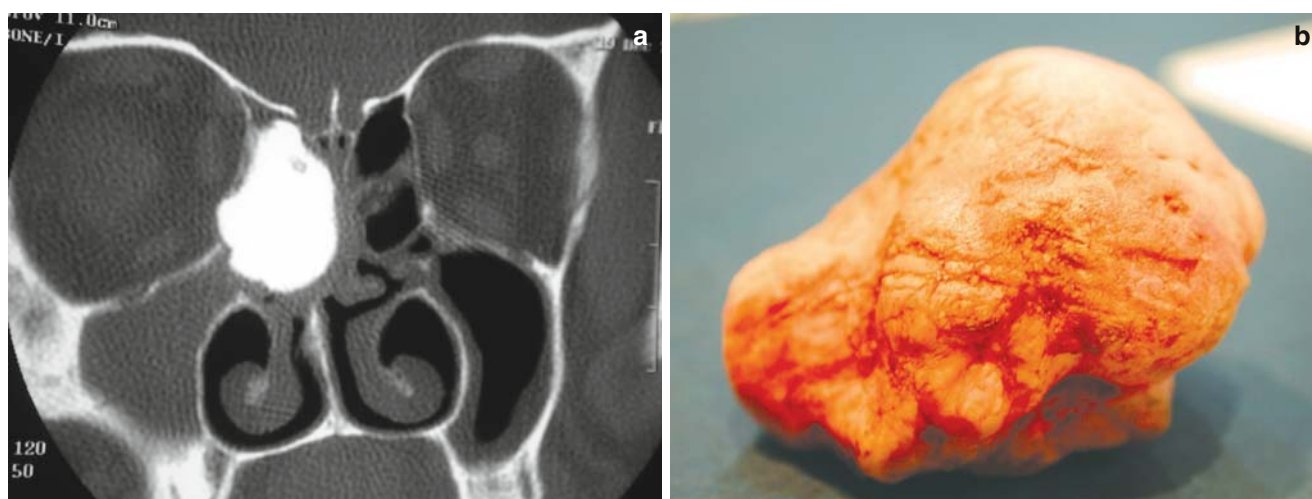


Fig. 2.13.6 Osteomas are common benign tumors of the sinuses. They occur most frequently in the frontoethmoid region. **(a)** Coronal CT showing osteoma in the right ethmoid area. **(b)** The specimen after removal

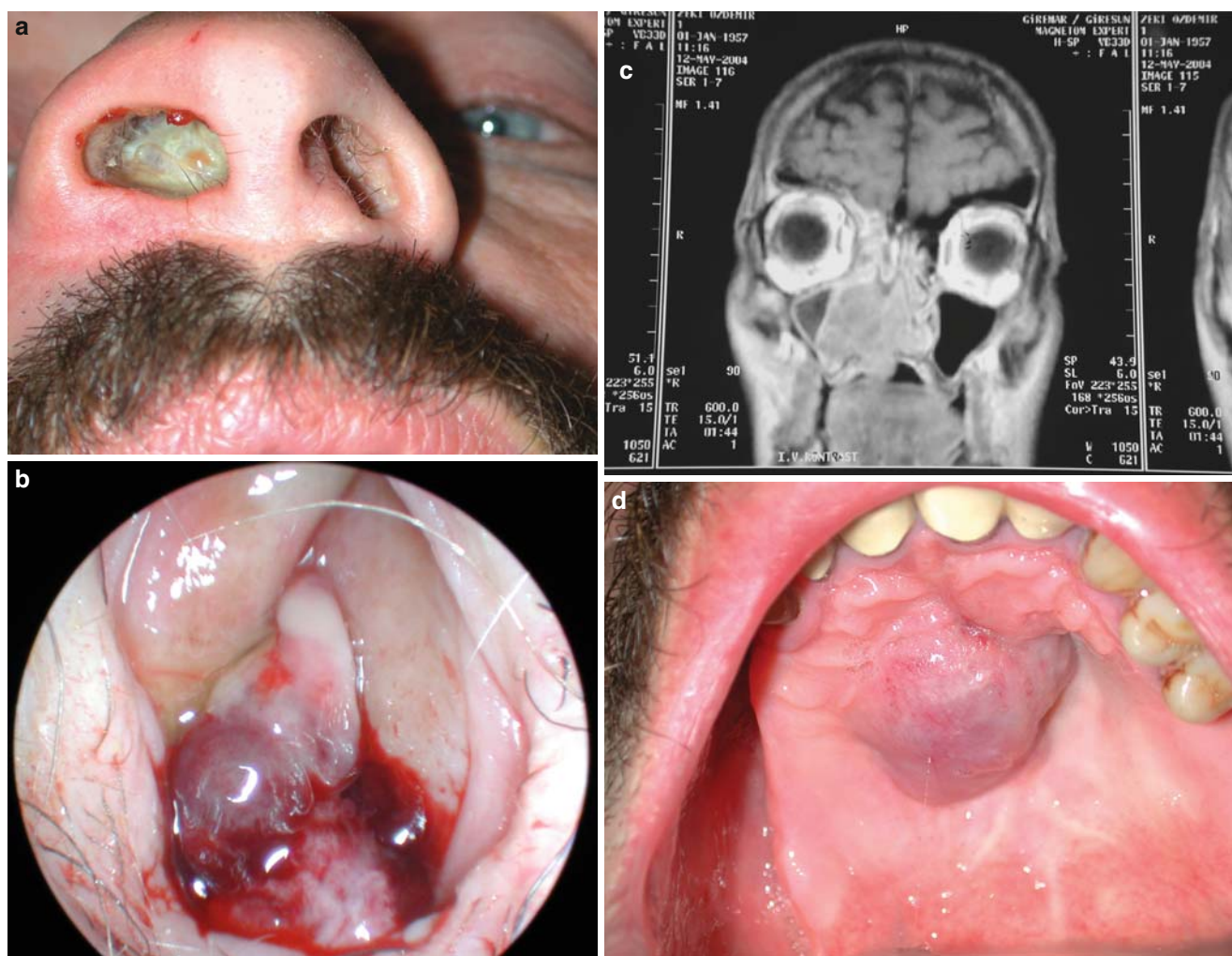


Fig. 2.13.7 Malignant melanoma in the nasal cavity. (a) Mass in the right nasal cavity. (b) Pigmented mass after cleaning of purulent material. (c) Coronal MR image shows mass filling the ethmoids and

maxillary sinus and extending to the palate. (d) Mass in the hard palate. Mass in the nasal cavity has eroded the palatal bone

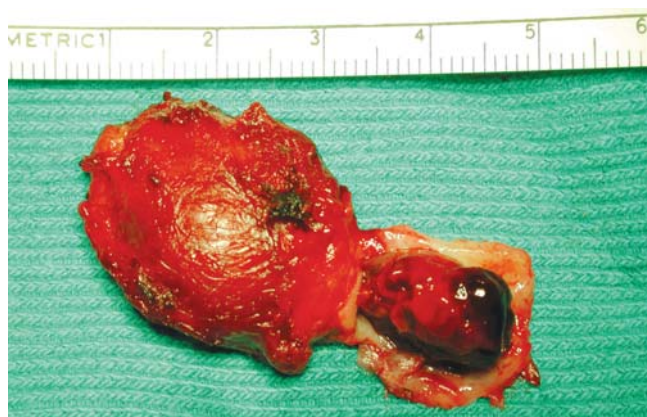


Fig. 2.13.8 Specimen of mucosal malignant melanoma in the nose after excision with safe margins. Malignant melanoma of the sinonasal tract comprises 1–2% of all melanomas. A pigmented polyp may be a malignant melanoma



Fig. 2.13.9 A unilateral mass in the nose that bleeds spontaneously should arouse the suspicion of malignancy. Esthesioneuroblastoma is an uncommon tumor that arises in the olfactory epithelium. These tumors very often invade the cranium. To show the intracranial extension, MR imaging is necessary

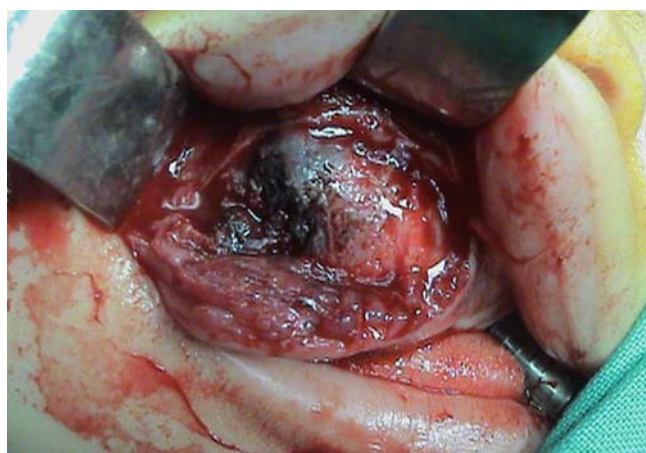


Fig. 2.13.10 Neuroblastomas are sarcomas of nervous system origin affecting mostly infants and children up to 10 years of age

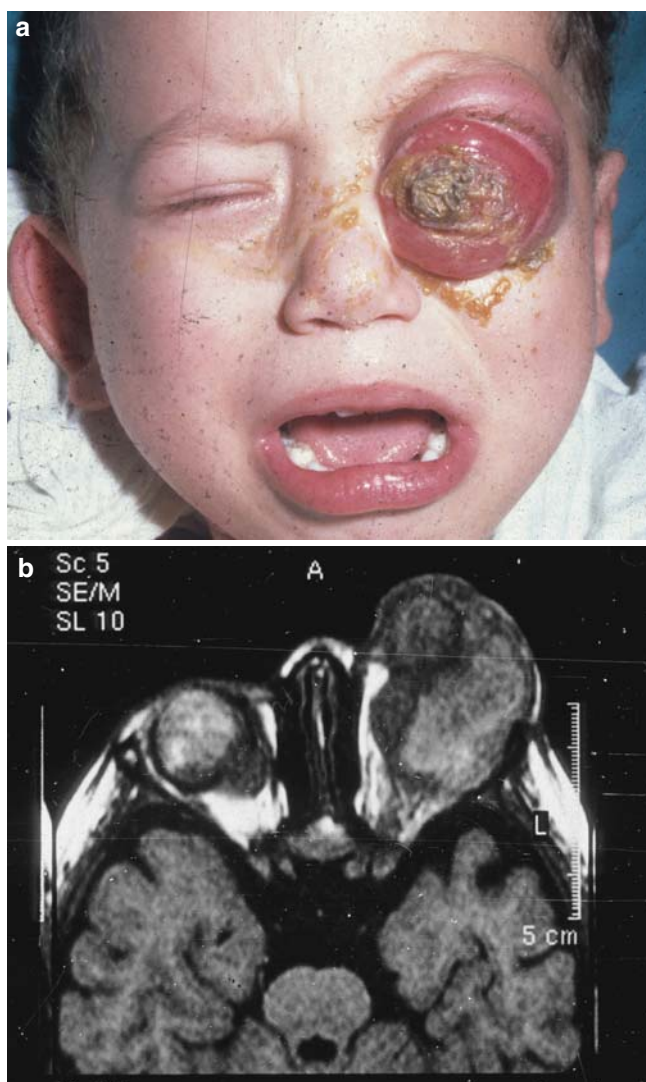


Fig. 2.13.11 (a) Retinoblastoma is the most frequent intraocular malignant tumor in childhood affecting mostly infants and children up to 3 years of age. **(b)** Axial MR image (courtesy of Dr. Kiratli)



Fig. 2.13.12 Lateral rhinotomy approach had been used for removal of tumors of the nasal cavity and paranasal sinuses. In the lateral rhinotomy approach, an incision is made along the nasofacial sulcus. This incision is extended into the nasal cavity along the nasolabial sulcus. The nasal flap is prepared and rotated upward and medially. Due to poor cosmetic results, this technique was replaced by other techniques



Fig. 2.13.13 Epidermoid carcinoma in the medial canthus of the right eye (courtesy of Dr. Kiratli)



Fig. 2.13.14 Epidermoid carcinoma in the left nasal vestibule



Fig. 2.13.15 Epidermoid carcinoma in the right nasal ala



Fig. 2.13.17 Epidermoid carcinoma that invades the nasal tip and nasal cavities



Fig. 2.13.16 Epidermoid carcinoma invading the columella and extending into both nasal cavities



Fig. 2.13.18 Epidermoid carcinoma filling the left gingivobuccal sulcus and invading the left maxillary sinus



Fig. 2.13.19 Total nose excision due to squamous cell carcinoma of the nose



Fig. 2.13.21 Excision of the tumor with exenteration of the eye



Fig. 2.13.20 Epidermoid carcinoma causing destruction of the face