

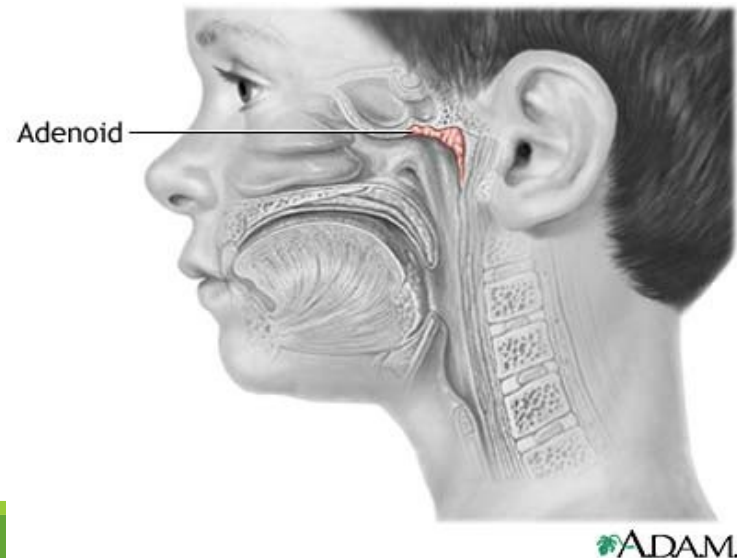
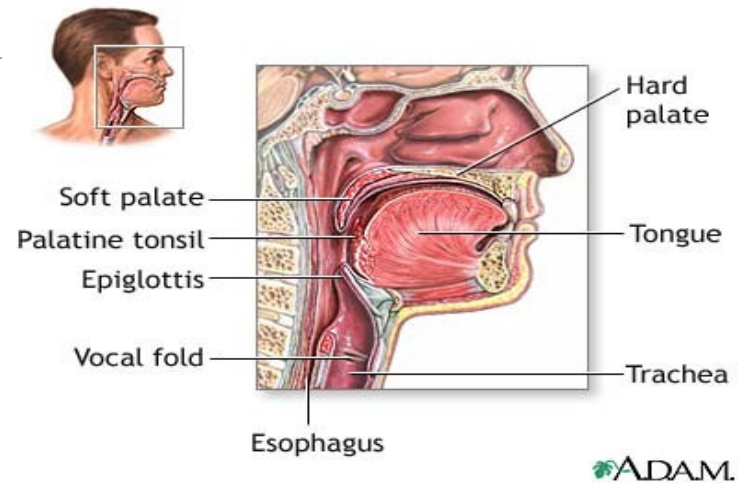
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فارنثريت

PHARYNGITIS

What is it?

- Inflammation of the Pharynx secondary to an infectious agent
- Most common infectious agents are Group A Streptococcus and various viral agents
- Often co-exists with tonsillitis



Etiology

- Strep.A
- Mycoplasma
- Strep.G
- Strep.C
- Corynebacterium diphtheriae
- Toxoplasmosis
- Gonorrhea
- Tularemia
- Rhinovirus
- Coronavirus
- Adenovirus
- CMV
- EBV
- HSV
- Enterovirus
- HIV

Acute Pharyngitis

Etiology

- Viral >90%
 - **Rhinovirus - common cold**
 - **Coronavirus - common cold**
 - **Adenovirus - pharyngoconjunctival fever; acute respiratory illness**
 - **Parainfluenza virus - common cold; croup**
 - **Coxsackievirus - herpangina**
 - **EBV - infectious mononucleosis**
 - **HIV**

Acute Pharyngitis

Etiology

- Bacterial
 - Group A beta-hemolytic streptococci (*S. pyogenes*)*
 - most common bacterial cause of pharyngitis
 - accounts for 15-30% of cases in children and 5-10% in adults.
 - *Mycoplasma pneumoniae*
 - *Arcanobacterium haemolyticum*
 - *Neisseria gonorrhea*
 - *Chlamydia pneumoniae*

PHARYNGITIS

HISTORY

- Classic symptoms → *Fever, throat pain, dysphagia*
 - *VIRAL* → Most likely concurrent URI symptoms of rhinorrhea, cough, hoarseness, conjunctivitis & ulcerative lesions
 - *STREP* → Look for associated headache, and/or abdominal pain
 - *Fever and throat pain are usually acute in onset*

PHARYNGITIS

- Physical Exam

- *VIRAL*

- EBV - White exudate covering erythematous pharynx and tonsils, cervical adenopathy,
 - *Subacute/chronic symptoms (fatigue/myalgias)*
 - *transmitted via infected saliva*
 - Adenovirus/Coxsackie - vesicles/ulcerative lesions present on pharynx or posterior soft palate
 - Also look for conjunctivitis

Epidemiology of Streptococcal Pharyngitis

- Spread by contact with respiratory secretions
- Peaks in winter and spring
- School age child (5-15 y)
- Communicability highest during acute infection
- Patient no longer contagious after 24 hours of antibiotics
- If hospitalized, droplet precautions needed until no longer contagious

PHARYNGITIS

- Physical Exam

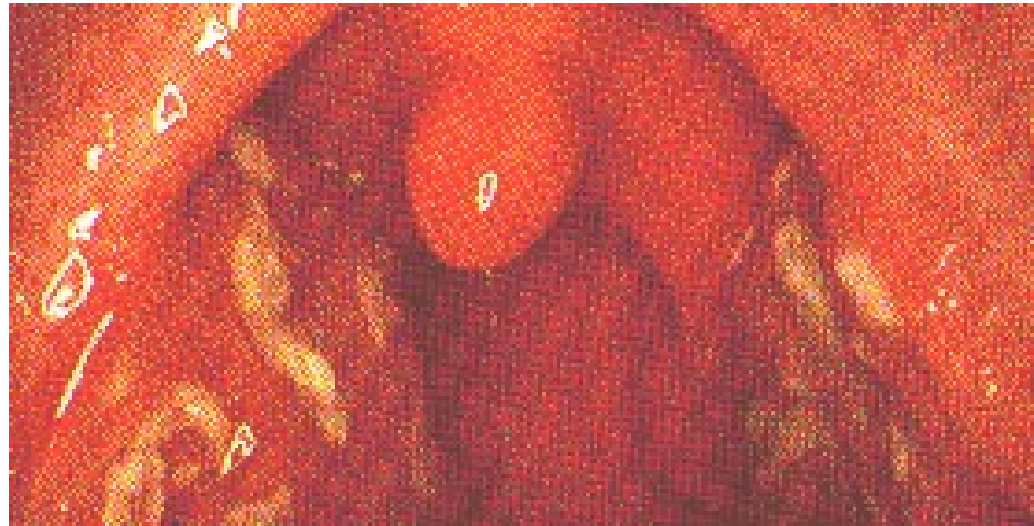
- *Bacterial*

- GAS – look for whitish exudate covering pharynx and tonsils
 - tender anterior cervical adenopathy
 - palatal/uvular petechiae
 - scarlatiniform rash covering torso and upper arms
- Spread via respiratory particle droplets – NO school attendance until 24 hours after initiation of appropriate antibiotic therapy
 - Absence of viral symptoms (rhinorrhea, cough, hoarseness)

Differential diagnosis of pharyngitis

Pharyngeal exudates:

- *S. pyogenes*
- *C. diphtheriae*
- *EBV*



Differential diagnosis of pharyngitis

Skin rash:

- *S. pyogenes*
- *HIV*
- *EBV*



Differential diagnosis of pharyngitis

Conjunctivitis:

- *Adenovirus*



Suppurative Complications of Group A Streptococcal Pharyngitis

Otitis media

Sinusitis

Peritonsillar and retropharyngeal abscesses

Suppurative cervical adenitis

Streptococcal Cervical Adenitis



Nonsuppurative Complications of Group A Streptococcus

Acute rheumatic fever

- follows only streptococcal pharyngitis (not group A strep skin infections)

Acute glomerulonephritis

- May follow pharyngitis or skin infection (pyoderma)
- Nephritogenic strains

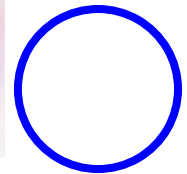
Pharyngitis



Infectious Mononucleosis



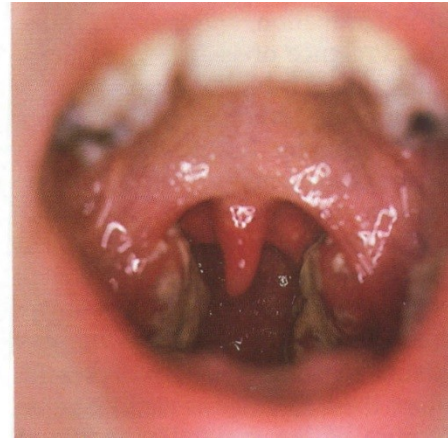
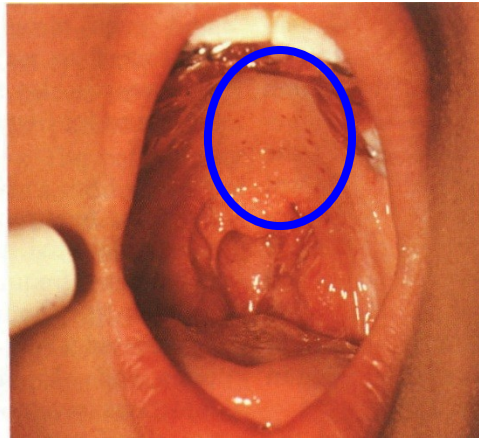
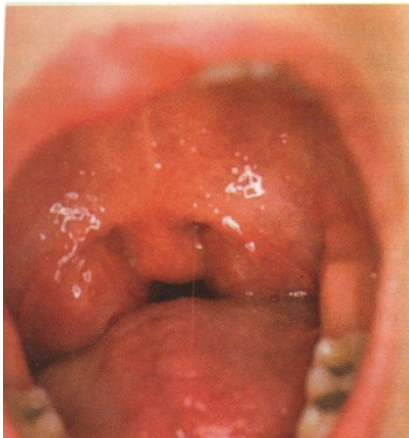
Herpangina



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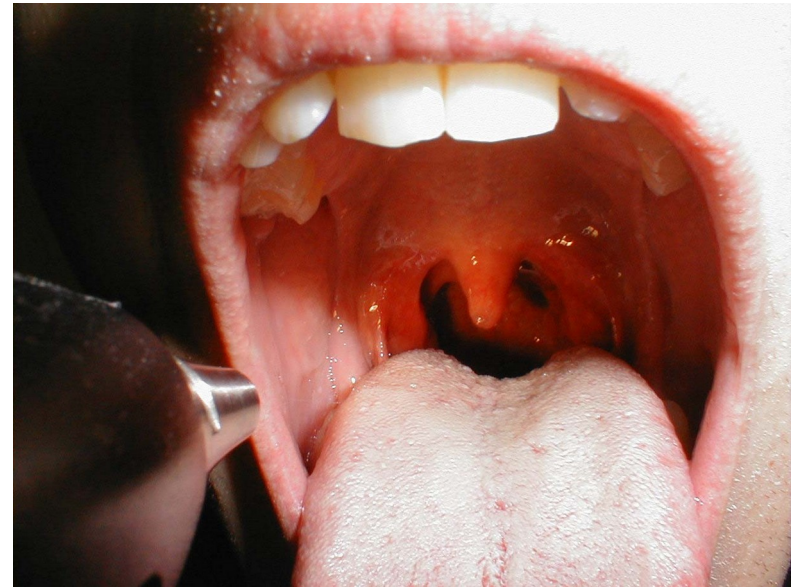
PHARYNGITIS



pharyngitis



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Scarlatiniform Rash



Clinical manifestation (Strep.)

Rapid onset

Headache

GI Symptoms

Sore throat

Erythma

Exudates

Palatine petechiae

Enlarged tonsils

Anterior cervical adenopathy
&Tender

Red& swollen uvula

Clinical manifestation (Viral)

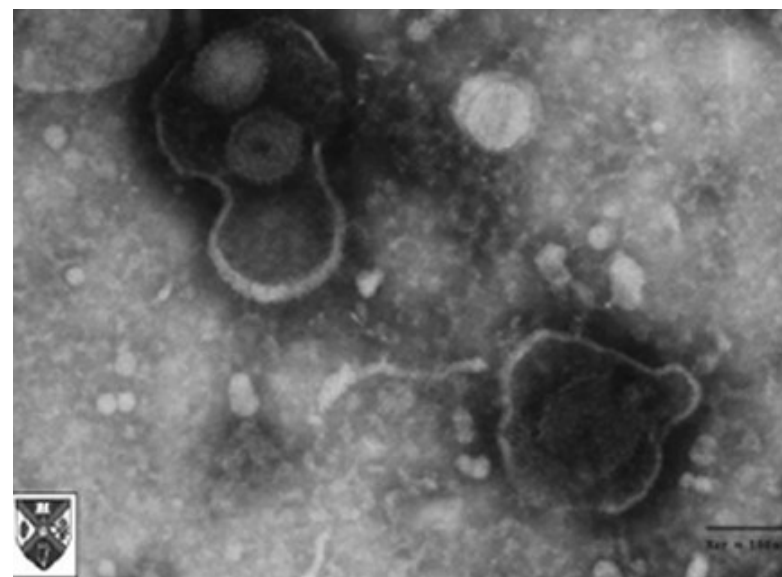
Gradual onset

Rhinorrhea

Cough

Diarrhea

Fever

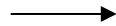


Clinical manifestation

- Vesiculation & Ulceration
Gingivostomatitis

→ HSV

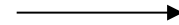
Coxsackievirus



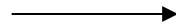
- Conjunctivitis

Adenovirus

- Gray-white fibrinous pseudomembrane
With marked cervical lymphadenopathy



Diphtheria

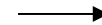


- Macular rash

Scarlet fever

- Hepatosplenomegally & Rash
& Fatigue & Cervical lymphadenitis

EBV



Diagnosis

- Strep:

Throat culture (Gold standard)

Rapid Strep. Antigen kits

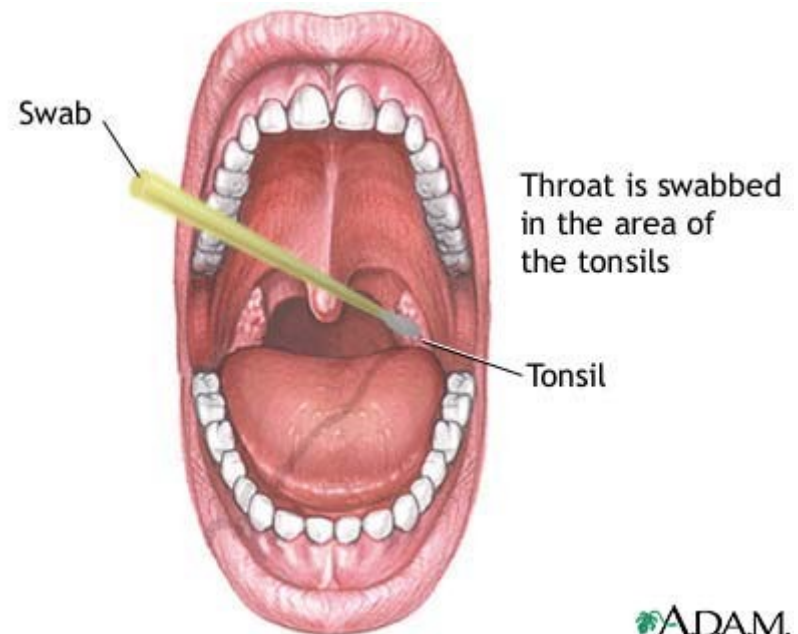
- Infectious Mono.:

CBC (Atypical lymphocytes)

Spot test (Positive slide agglutination)

- Mycoplasma:

Cold agglutination test



Differential diagnosis

- Retropharyngeal abscesses
- Peritonsillar abscesses
- Ludwig angina
- Epiglottitis
- Thrush
- Autoimmune ulceration
- Kawasaki

Treatment

(Antibiotic ,Acetaminophen ,Warm salt gargling)

- Strep: *Penicillin* , Erythromycin , Azithromycin

- Carrier of strep:

Clindamycin , Amoxicillin clavulanic

- Retropharyngeal abscesses:

Drainage + Antibiotics

- Peritonsillar abscesses:

penicillin + Aspiration

Recurrent pharyngitis

- Etiology: Nonpenicillin treatment ,Different strain ,Another cause pharyngitis
- Treatment:

Tonsilectomy

if

Culture positive, severe GABHS more than
7 times during previous year

or

5 times each year during two previous year

Benefit of treatment of Strep. Pharyngitis

1-Prevention of ARF if treatment started within 9 days of illness

2-Reduce symptoms

3-Prevent local suppurative complications

BUT

Does not prevent the development of the post streptococcal sequel of acute glomerulonephritis

Antibiotic started immediately with symptomatic pharyngitis and positive Rapid test (Without culture)

1-Clinical diagnosis of scarlet fever

2-Household contact with documented strep. Pharyngitis

3-Past history of ARF

4-Recent history of ARF in a family member

PHARYNGITIS

- LAB AIDS

- Rapid strep antigen → detects GAS antigen
 - Tonsillar swab → 3-5 minutes to perform
 - 95% specificity, 90-93% sensitivity
- GAS Throat culture → “gold standard”
 - >95% sensitivity
- Mono Spot → serologic test for EBV heterophile Ab
- EBV Ab titers → detect serum levels of EBV IgM/IgG

PHARYNGITIS

- Treatment

- VIRAL – Supportive care only – Analgesics, Antipyretics, Fluids
- No strong evidence supporting use of oral or intramuscular corticosteroids for pain relief → few studies show transient relief within first 12–24 hrs after administration
- EBV – infectious mononucleosis
 - activity restrictions – mortality in these pts most commonly associated with abdominal trauma and splenic rupture

PHARYNGITIS

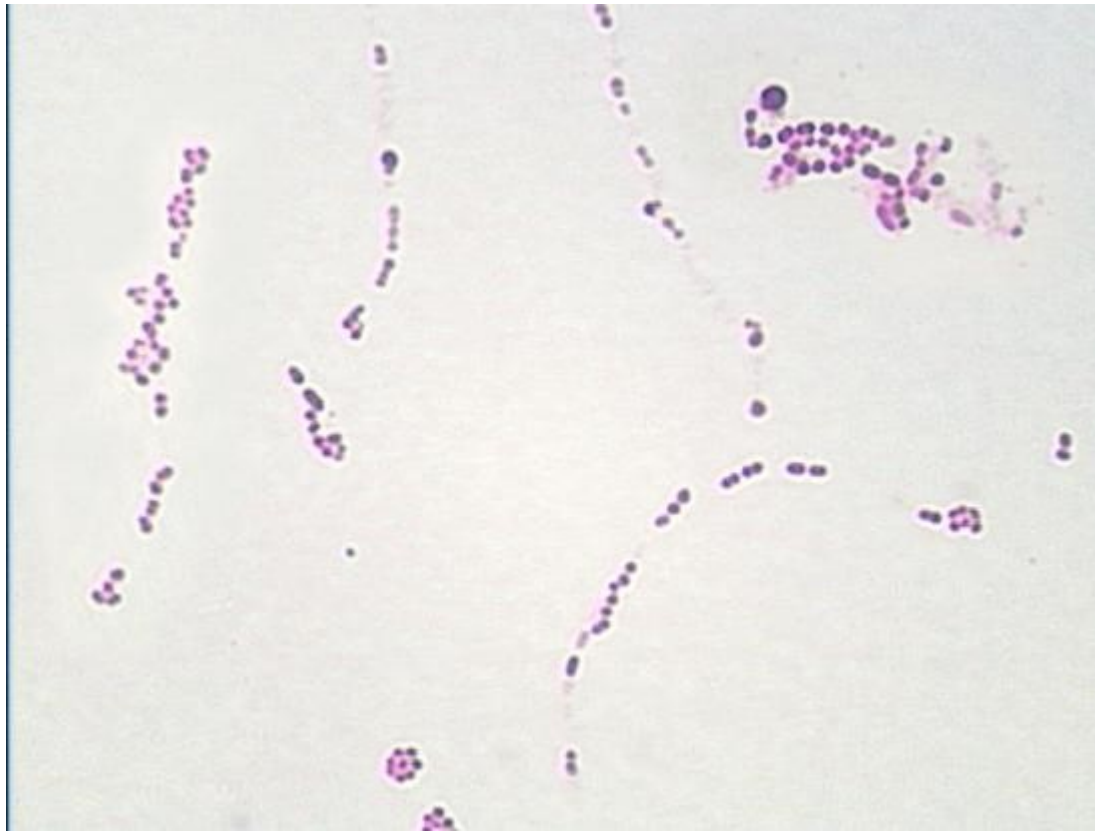
- Treatment → Do so to prevent *ARF*
Rheumatic Fever)

(Acute

- GAS →

- ✓ Oral PCN – treatment of choice
 - ✓ 10 day course of therapy
- ✓ IM Benzathine PCN G – 1.2 million units x 1
- ✓ Azithromycin, Clindamycin, or 1st generation cephalosporins for PCN allergy

Group A Streptococcus

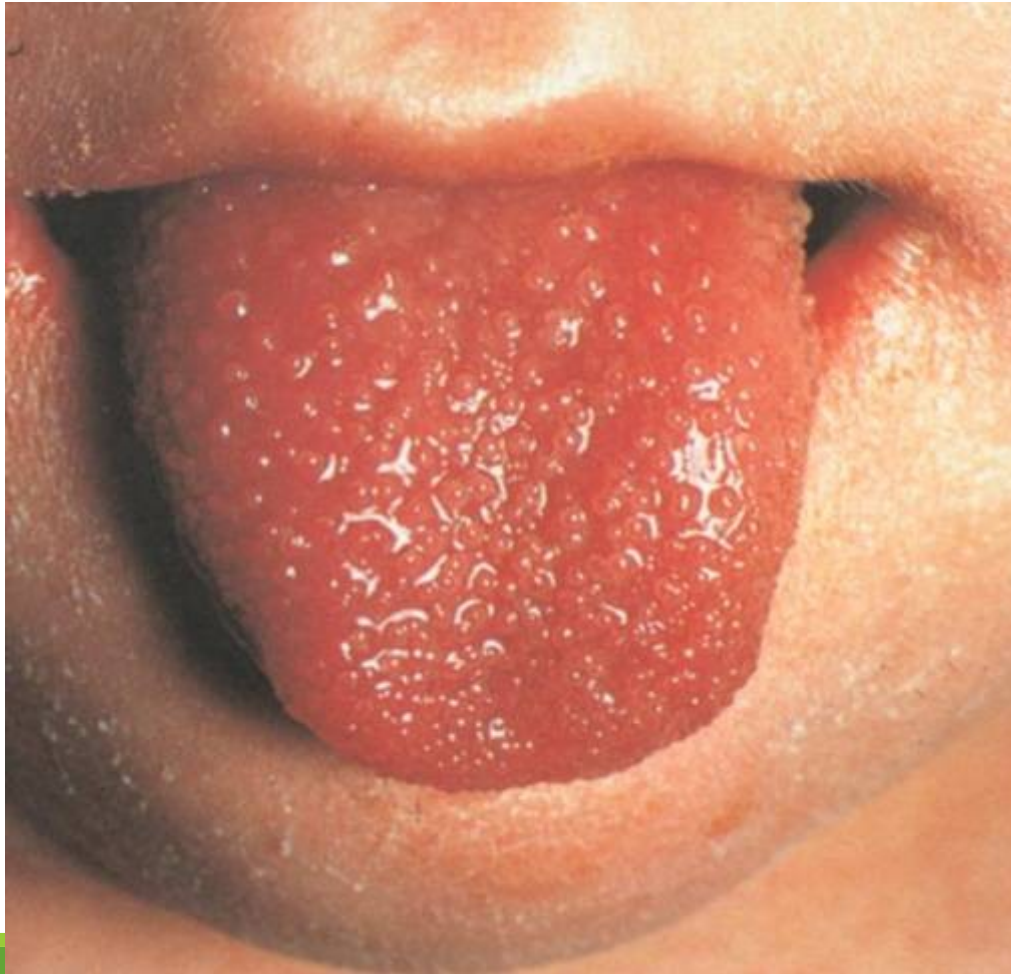


Group A Beta Hemolytic Streptococcus





Strawberry Tongue in Scarlet Fever



Scarlet Fever

Occurs most commonly in association with pharyngitis

- Strawberry tongue
- Rash
 - Generalized fine, sandpapery scarlet erythema with accentuation in skin folds (Pastia's lines)
 - Circumoral pallor
 - Palms and soles spared
- Treatment same as strep pharyngitis

Rash of Scarlet Fever



Acute Rheumatic Fever

Immune mediated - ?humoral

Diagnosis by Jones criteria

- 5 major criteria

- Carditis

- Polyarthrititis (migratory)

- Sydenham's chorea

- muscular spasms, incoordination, weakness

- Subcutaneous nodules

- painless, firm, near bony prominences

- Erythema marginatum

Erythema Marginatum



Acute Rheumatic Fever

- Minor manifestations
 - Clinical Findings
 - arthralgia
 - fever
 - Laboratory Findings
 - Elevated acute phase reactants
 - erythrocyte sedimentation rate
 - C-reactive protein
 - Prolonged P-R interval on EKG

Acute Rheumatic Fever

Supporting evidence of antecedent group A streptococcal infection

- Positive throat culture or rapid streptococcal antigen test
- Elevated or rising streptococcal antibody titer
 - antistreptolysin O (ASO), antiDNase B

If evidence of prior group A streptococcal infection, 2 major or one major and 2 minor manifestations indicates high probability of ARF

Acute Rheumatic Fever

Therapy

- Goal: decrease inflammation, fever and toxicity and control heart failure
- Treatment may include anti-inflammatory agents and steroids depending on severity of illness

Poststreptococcal Glomerulonephritis

Develops about 10 days after pharyngitis

Immune mediated damage to the kidney that results in renal dysfunction

Nephritogenic strain of *S. pyogenes*

Poststreptococcal Glomerulonephritis

Clinical Presentation

- Edema, hypertension, and smoky or rusty colored urine
- Pallor, lethargy, malaise, weakness, anorexia, headache and dull back pain
- Fever not prominent

Laboratory Findings

- Anemia, hematuria, proteinuria
- Urinalysis with RBCs, WBCs and casts

Poststreptococcal Glomerulonephritis

Diagnosis

- Clinical history, physical findings, and confirmatory evidence of antecedent streptococcal infection (ASO or anti-DNase B)

Therapy

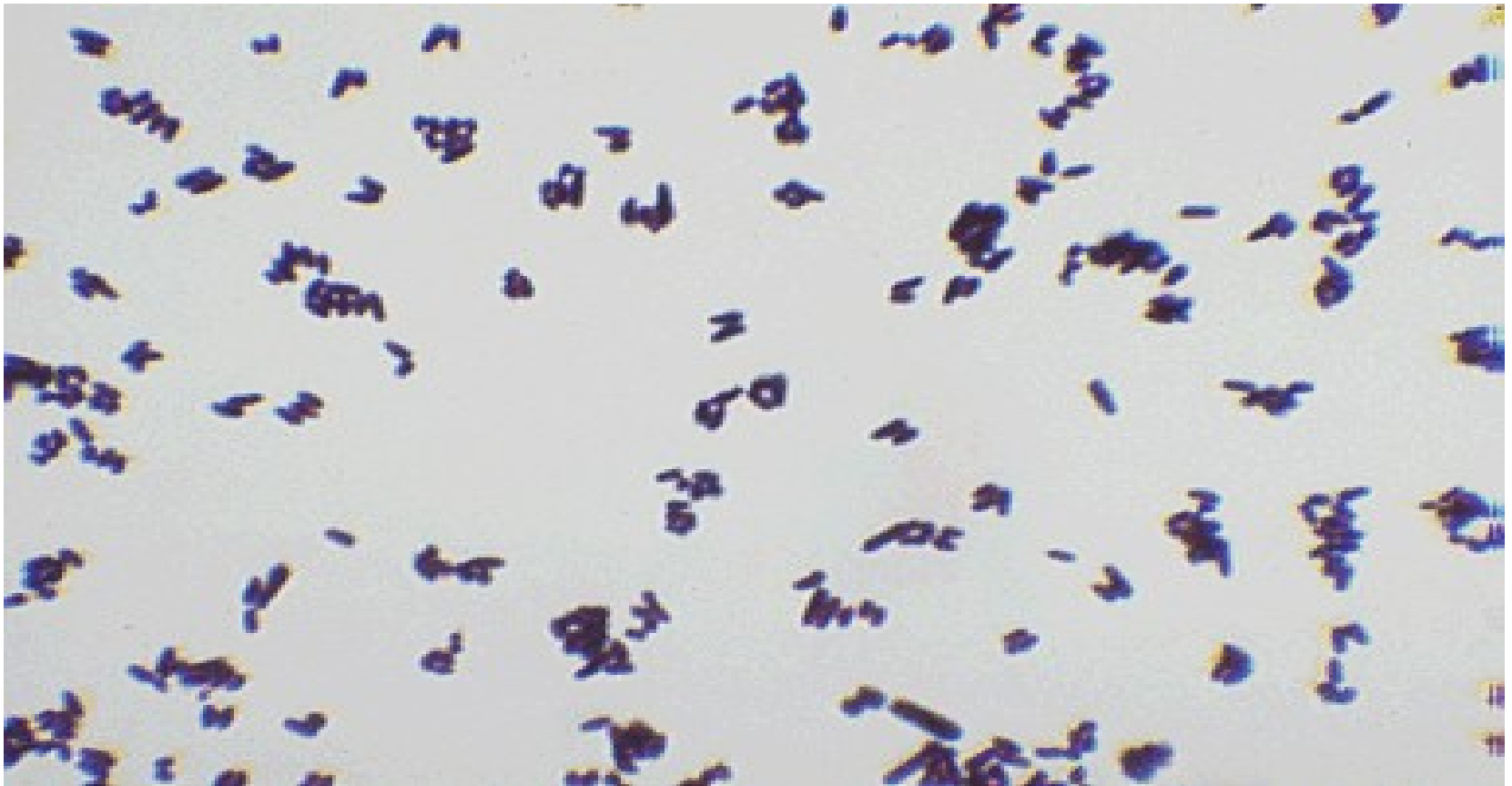
- Penicillin to eradicate the nephritogenic streptococci (erythromycin if allergic)
- Supportive care of complications

Diphtheria

Etiologic agent: Corynebacterium diphtheria

- Extremely rare, occurs primarily in unimmunized patients
- Gram positive rod
- nonspore forming
- strains may be toxigenic or nontoxigenic
 - exotoxin required for disease

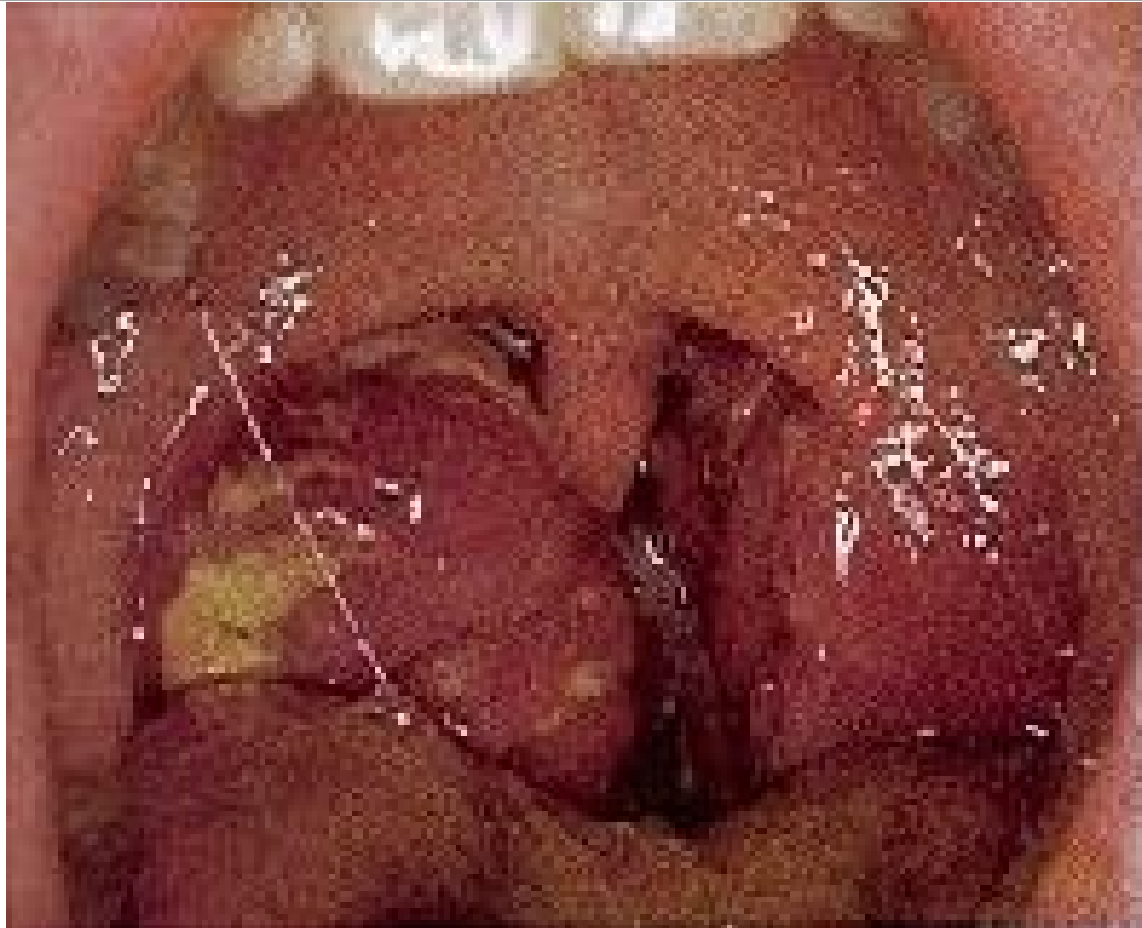
Corynebacterium Diphtheriae



TONSILLITIS

- Inflammation/Infection of the tonsils
 - Palatine tonsils → visible during oral exam
 - Also have pharyngeal tonsils (adenoids) and lingual tonsils
- History → sore throat, fever, otalgia, dysphagia
- Physical Exam → whitish plaques, enlarged/tender cervical adenopathy
- Etiology → GAS, EBV – less commonly HSV
- Treatment → same as for pharyngitis

TONSILLITIS



TONSILLITIS



LARYNGITIS

Inflammation of the mucous membranes covering the larynx with accompanied edema of the vocal cords

- History → sore throat, dysphonia (hoarseness) or loss of voice, cough, possible low-grade fever
- Physical Exam →
 - cannot directly visualize larynx on standard PE
 - must use fiberoptic laryngoscopy (not usually necessary)

LARYNGITIS

- ETIOLOGY →

- Acute [<3wks duration]- Think infectious → most commonly viral – symptoms most commonly resolve in 7-10 days
- Chronic [>3wks duration]- Inhalation of irritant fumes, vocal misuse, GERD, smokers
- Treatment → symptomatic care → complete voice rest, avoid exposure to insulting agent, anti-reflux therapy
 - Prevailing data does NOT support the use of corticosteroids for symptomatic relief

PERITONSILLAR ABSCESS

- Accumulation of pus in the tonsillar fossa → thought to be an infectious complication of inappropriately treated pharyngitis/tonsillitis
- History →
 - Antecedent sore throat 1-2 wks prior - progressively worsens
 - Dysphagia
 - High fever
 - Ipsilateral throat, ear & possibly neck pain
- Physical Exam →
 - Trismus - 67% of cases
 - muffled voice (“Hot Potato”)
 - Drooling &/or fetid breath
 - look for unilateral mass in the supratonsillar area with possible uvula deviation
 - fluctuant upon palpation

PERITONSILLAR ABSCESS

- Etiology → 90% of aspirated cultures grow bacterial pathogens
 - ✓ GAS – most common (approximately 30% of cases)
 - ✓ Staphylococcus aureus
 - ✓ Anaerobes – most commonly Peptostreptococcal microbes
- Treatment →
 - ✓ Prompt ENT consultation for needle aspiration (*always send cultures) or possible surgical drainage
 - ✓ Systemic abx – *usually Clindamycin and a β -Lactam or 1st generation cephalosporin*
 - ✓ Surgical tonsillectomy if:
 - 1) No improvement in 48 hours
 - 2) H/O recurrent abscesses – 3 or more (controversial)





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Clinical Features

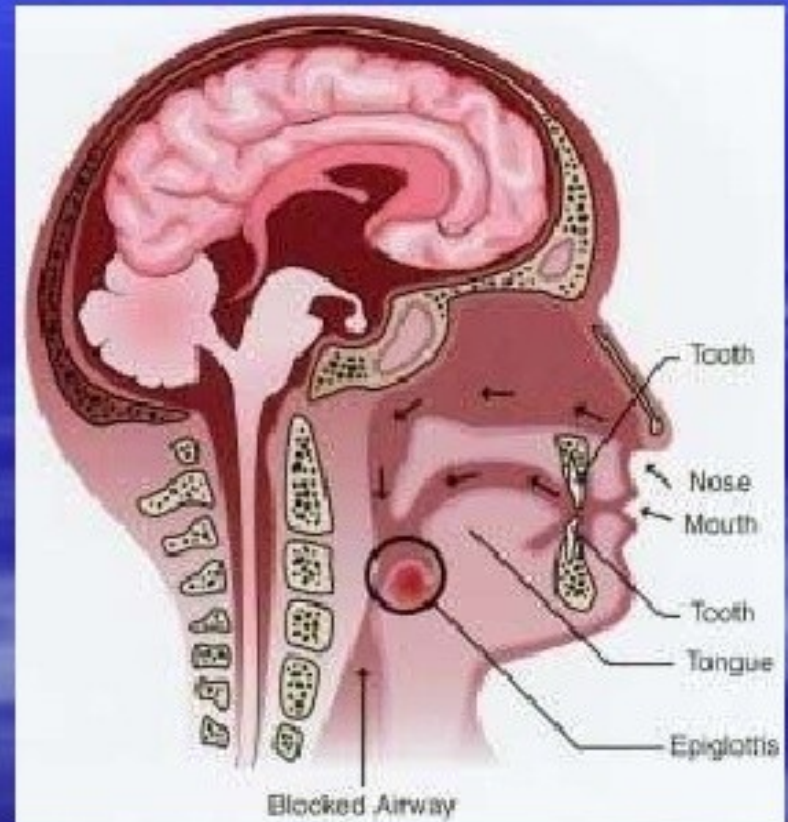
Bacterial cellulitis of the epiglottis (supraglottis) and/or surrounding tissue.

- *Haemophilus influenzae* type b (HiB) most likely pathogen
- *H. parainfluenzae* and streptococci are also reported.

Average age of onset: 1–5 years old.

Sudden onset: sore throat, fever, head forwardly extended, usually with drooling.

Resps: delicate, little movement of head. May hear stridor.



Causal organisms in children and adults

include *Streptococcus pneumoniae*, *Staphylococcus aureus*, nontypeable *H. influenzae*, *Haemophilus parainfluenzae*, β -hemolytic streptococci, *Branhamella catarrhalis*, and *Klebsiella pneumoniae*. *H. influenzae* type B is still a cause in adults and unvaccinated children.

Bacteria that have colonized the nasopharynx spread locally to cause supraglottic cellulitis with marked inflammation of the epiglottis, vallecula, aryepiglottic folds, arytenoids, and laryngeal ventricles. With *H. influenzae* type B, infection may spread hematogenously.

The inflamed supraglottic structures mechanically obstruct the airway, increasing the work of breathing, ultimately causing respiratory failure. Clearance of inflammatory secretions is also impaired.



Symptoms and Signs

In children, sore throat, odynophagia, and dysphagia develop abruptly.

Fatal asphyxia may occur within a few hours of onset. Drooling is very common. Additionally, the child has signs of toxicity (poor or absent eye contact, failure to recognize parents, cyanosis, irritability, inability to be consoled or distracted) and is febrile and anxious.

Dyspnea, tachypnea, and inspiratory stridor may be present, often causing the child to sit upright, lean forward, and hyperextend the neck with the jaw thrust forward and mouth open in an effort to enhance air exchange (tripod position). Relinquishing this position may herald respiratory failure. Suprasternal, supraclavicular, and subcostal inspiratory retractions may be present.



Symptoms of Epiglottitis



High fever



Hoarse voice



Anxiety and
Restlessness



In adults, symptoms are similar to those of children, including sore throat, fever, dysphagia, and drooling, but peak symptoms usually take > 24 h to develop.

Because of the larger diameter of the adult airway, obstruction is less common and less fulminant. Often, there is no visible oropharyngeal inflammation.

However, severe throat pain with a normal-appearing pharynx raises suspicion of epiglottitis. A delay in diagnosis and treatment increases the risk of airway obstruction and death.

Diagnosis

Direct inspection (typically in operating room)

X-ray in milder cases with low suspicion

Epiglottitis is suspected in patients with severe sore throat and no pharyngitis and also in patients with sore throat and inspiratory stridor.

Stridor in children may also result from croup (viral laryngotracheal bronchitis—see Table: Differentiating Epiglottitis From Croup), bacterial tracheitis, and airway foreign body. The tripod position may also occur with peritonsillar or retropharyngeal abscess.

Epiglottitis vs Croup

Feature	Epiglottitis	Croup*
Onset	Acute and fulminant	More gradual
Age	Commonly, 2–8 yr (if not vaccinated against Haemophilus influenzae type B) and adults	Commonly, 6–36 mo
Barking cough	Uncommon	Characteristic
Epiglottis	Edematous and cherry red	May be erythematous
Neck x-ray findings	Enlarged epiglottis (thumb sign) and distention of the hypopharynx	Subglottic narrowing (steeple sign) and a normal-sized epiglottis
* Also called viral laryngotracheal bronchitis.		



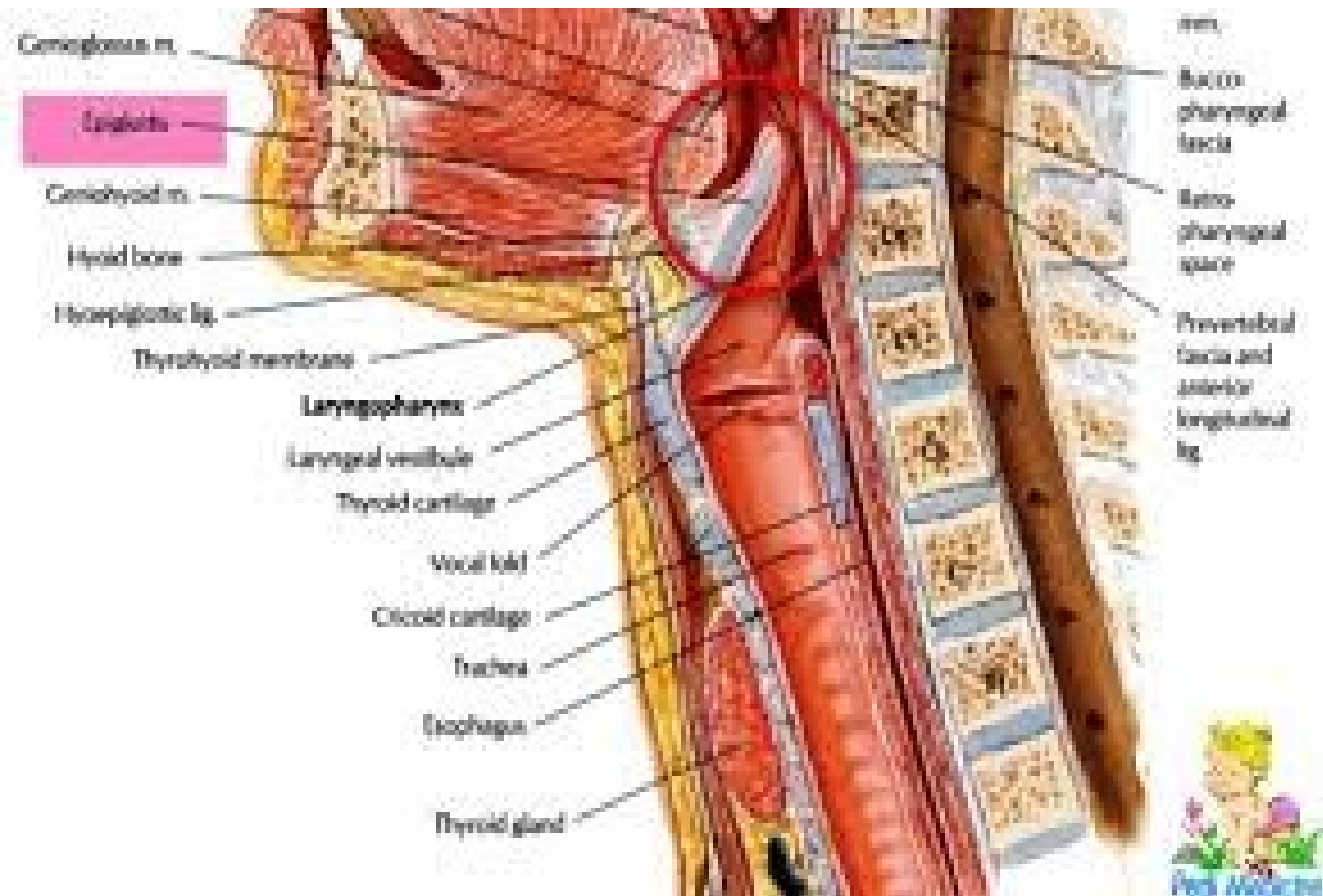
The patient is hospitalized if epiglottitis is suspected.

Diagnosis requires direct examination, usually with flexible fiberoptic laryngoscopy. (CAUTION: *Examination of the pharynx and larynx may precipitate complete respiratory obstruction in children, and the pharynx and larynx should not be directly examined except in the operating room, where the most advanced airway intervention is available.*)

plain x-rays may be helpful, a child with stridor should not be transported to the x-ray suite.

Direct laryngoscopy that reveals a beefy-red, stiff, edematous epiglottitis is diagnostic. Cultures from the supraglottic tissues and blood can then be taken to search for the causative organism.

Adults may, in some cases, safely undergo flexible fiberoptic laryngoscopy.









W1

Pearls & Pitfalls

- Examination of the pharynx or larynx in children with epiglottitis and stridor may precipitate complete airway obstruction.

Treatment

Airway

Antibiotics (eg, ceftriaxone)

In children with stridor, any intervention that could be upsetting (and thus could trigger airway obstruction) should be avoided until an airway is established. In children with epiglottitis, the airway must be secured immediately. Securing the airway can be quite difficult and should, if possible, be done by experienced

personnel in the operating room.

An endotracheal tube is usually required until the patient has been stabilized for 24 to 48 h (usual total intubation time is < 60 h).

Alternatively, a tracheotomy is done

If respiratory arrest

occurs before an airway is established, bag-mask ventilation may be a life-saving temporary measure. For emergency care of children with epiglottitis, each institution should have a protocol that involves critical care, otolaryngology, anesthesia, and pediatrics.

Adults whose airway is severely obstructed can be endotracheally intubated during flexible fiberoptic laryngoscopy. Other adults may not require immediate intubation but should be observed for airway compromise in an ICU with an intubation set and cricothyrotomy tray at the bedside.

A β -lactamase-resistant antibiotic, such as ceftriaxone 50 to 75 mg/kg IV once/day (maximum 2 g), should be used empirically, pending culture and sensitivity test results.

Epiglottitis caused by *H. influenzae* type B can be effectively prevented with the *H. influenzae* type B (HiB) conjugate vaccine.

Key Points

The incidence of epiglottitis has decreased significantly, particularly in children, because of widespread vaccination against the most common cause, *Haemophilus influenzae* type B.

Stridor, as well as sore throat with a normal-appearing pharynx, are important clues.

Examination of the pharynx or larynx in children with epiglottitis and stridor may precipitate complete airway obstruction.

If the diagnosis is suspected, do flexible **fiberoptic laryngoscopy** in the operating room; reserve imaging studies for cases with very low suspicion.

Children typically should have their airway secured by tracheal intubation; adults often can be observed for signs of airway compromise.

Give a β -lactamase-resistant antibiotic, such as ceftriaxone.

