Case scenario

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5 yrs old with mild fever,dysphasia, left sided neck swelling. There is a patch on tonsil which bleeds on removal, name the disease and its complication. Discuss the d/d ,complication and management

- **Discussion:** 5 yr with unknown immune status has fever, neck swelling, membrane on tosil which bleeds is likely to membranous tonsillitis. The degree of fever being mild, neck swelling ,bleeding membranous tonsiltis,dysphasia with unknown immune status is likely to have diphtheria,eventhough other causes of membranous tonsillitis are to be considered. The characteristic adherent membrane, extension beyond the faucial area, dysphagia, and relative lack of fever help differentiate diphtheria from exudative pharyngitis caused by *Streptococcus pyogenes* or Epstein-Barr virus
- **Life-threatening complications** of respiratory diphtheria include upper airway obstruction caused byextensive membrane formation; myocarditis, which often is associated with heart block;and cranial and peripheral neuropathies. Palatal palsy, characterized by nasal speech, frequently occurs in pharyngeal diphtheria.

D/D

STREPTOCCAL GR A PHARYNGITIS	The onset of streptococcal pharyngitis is often rapid, with prominent sore throat and fever in the absence of cough. Headache and gastrointestinal symptoms (abdominal pain, vomiting) are common. The pharynx is red, and the tonsils are enlarged and classically covered with a yellow, blood-tinged exudate. There may be petechiae or “doughnut” lesions on the soft palate and posterior pharynx, and the uvula may be red, stippled, and swollen. The anterior cervical lymph nodes are enlarged and tender. The incubation period is 2-5 days. Some patients demonstrate the additional stigmata of scarlet fever: circumoral pallor, strawberry tongue, and a red, finely papular rash that feels like sandpaper and resembles sunburn with goose pimples The specificity of rapid tests to detect group A streptococcal antigen is high, so if a rapid test is positive, throat culture is unnecessary and appropriate treatment is indicated. Because rapid tests are generally less sensitive than culture, confirming a negative rapid test with a throat culture has been recommended, especially if the clinical suspicion of GABHS is high. Special culture media and a prolonged incubation are required to detect *A. haemolyticum.* Viral cultures are often unavailable and are generally too expensive and slow to be clinically useful. Viral polymerase chain reaction (PCR) is more rapid and may be useful but is not always necessary.

EBV PHARYNGITIS/VIRAL	▪ The onset of viral pharyngitis may be more gradual, and symptoms more often include rhinorrhea, cough, and diarrhea. A viral etiology is suggested by the presence of conjunctivitis, coryza, hoarseness, and cough. ▪ Adenovirus pharyngitis can feature concurrent conjunctivitis and fever (pharyngoconjunctival fever;. Coxsackievirus pharyngitis can produce small (1-2 mm) grayish vesicles and punched-out ulcers in the posterior pharynx (herpangina), or small (3-6 mm) yellowish-white nodules in the posterior pharynx (acute lymphonodular pharyngitis;. ▪ In EBV pharyngitis, there may be prominent tonsillar enlargement with exudate, cervical lymphadenitis, hepatosplenomegaly, rash, and generalized fatigue as part of the infectious mononucleosis syndrome. Primary HSV infections in young children often manifest as high fever and gingivostomatitis, but pharyngitis may be present
CANDIDIASIS	▪ It is characterized by pearly white, curdish material visible on the tongue, palate, and buccal mucosa. Oral thrush may be asymptomatic or can cause pain, fussiness, and decreased feeding, leading to inadequate nutritional intake and dehydration. It is uncommon after 1 yr of age but can occur in older children treated with antibiotics. Persistent or recurrent thrush with no obvious predisposing reason, such as recent antibiotic treatment, warrants investigation of an underlying immunodeficiency, especially vertically transmitted HIV infection.
VINCENTS ANGINA	▪ **acute necrotizing ulcerative gingivitis** or **trench mouth,** is an acute, fulminating, mixed anaerobic bacterial-spirochetal infection of the gingival margin and floor of the mouth. It is characterized by gingival pain, foul breath, and pseudomembrane formation. *Fusobacterium* Necrotizing gingivitis (Vincent stomatitis) | *Prevotella melaninogenica* ▪
LEMERIE'S	▪ is a suppurative infection of the lateral pharyngeal space, of apparent increasing prevalence, that often begins as pharyngitis It may complicate Epstein Barr Virus or other infections of the pharynx. It usually manifests as a unilateral septic thrombophlebitis of the jugular venous system with septic pulmonary embolization. Clinical signs include unilateral painful neck swelling, trismus, and dysphagia culminating with signs of sepsis and respiratory distress. *Fusobacterium necrophorum* is the most commonly isolated organism, although polymicrobial infection may occur
CROUP	▪ hoarse voice, coryza, normal to moderately inflamed pharynx, and a slightly increased respiratory rate. Patients

	vary substantially in their degrees of respiratory distress. Rarely, the upper airway obstruction progresses and is accompanied by an increasing respiratory rate; nasal flaring; suprasternal, infrasternal, and intercostal retractions; and continuous stridor. Croup is a disease of the upper airway, and alveolar gas exchange is usually normal
EPIGLOTTIS	▪ an acute rapidly progressive and potentially fulminating course of high fever, sore throat, dyspnea, and rapidly progressing respiratory obstruction. The degree of respiratory distress at presentation is variable. The initial lack of respiratory distress can deceive the unwary clinician; respiratory distress can also be the 1st manifestation. Often, the otherwise healthy child suddenly develops a sore throat and fever. Within a matter of hours, the patient appears toxic, swallowing is difficult, and breathing is labored. Drooling is usually present and the neck is hyperextended in an attempt to maintain the airway.
STAPH TRACHEITIS	▪ child has a brassy cough, apparently as part of a viral laryngotracheobronchitis. High fever and "toxicity" with respiratory distress can occur immediately or after a few days of apparent improvement. The patient can lie flat, does not drool, and does not have the dysphagia associated with epiglottitis. The usual treatment for croup (racemic epinephrine) is ineffective.

COMPLICATIONS

- Respiratory tract obstruction by pseudomembranes may require bronchoscopy or intubation and mechanical ventilation. Two other tissues usually remote from sites of *C. diphtheriae* infection can be significantly affected by **diphtheritic toxin:** the heart and the nervous system
- **Life-threatening complications** of respiratory diphtheria include upper airway obstruction caused by extensive membrane formation; myocarditis, which often is associated with heart block;and cranial and peripheral neuropathies. Palatal palsy, characterized by nasal speech, frequently occurs in pharyngeal diphtheria

Toxic Cardiomyopathy

- Toxic cardiomyopathy occurs in 10-25% of patients with respiratory diphtheria and is responsible for 50-60% of deaths. Subtle signs of myocarditis can be detected in most patients, especially the elderly, but the risk for significant complications correlates directly with the extent and severity of exudative local oropharyngeal disease as well as delay in administration of antitoxin.
- The 1st evidence of cardiac toxicity characteristically occurs during the 2nd and 3rd weeks of illness as the pharyngeal disease improves but can appear acutely as early as the 1st wk of illness, a poor prognostic sign, or insidiously as late as the 6th wk. **Tachycardia** out of

proportion to fever is common and may be evidence of cardiac toxicity or autonomic nervous system dysfunction. A prolonged PR interval and changes in the ST-T wave on an electrocardiographic tracing are relatively frequent findings; dilated and hypertrophic cardiomyopathy detected by echocardiogram has been described.

- Single or progressive cardiac **dysrhythmias** can occur, including 1st-, 2nd-, and 3rd-degree heart block. Temporary transvenous pacing may improve outcomes. Atrioventricular dissociation and ventricular tachycardia are also described, the latter having a high associated mortality. Heart failure may appear insidiously or acutely. Elevation of the serum aspartate aminotransferase concentration closely parallels the severity of myonecrosis. Severe dysrhythmia portends death.
- Histologic postmortem findings are variable: little or diffuse myonecrosis with acute inflammatory response. Recovery from toxic myocardiopathy is usually complete, although survivors of more severe dysrhythmias can have permanent conduction defects.

Toxic Neuropathy

- Neurologic complications parallel the severity of primary infection and are multiphasic in onset. Acutely or 2-3 wk after onset of oropharyngeal inflammation, it is common for hypesthesia and local **paralysis** of the soft palate to occur. Weakness of the posterior pharyngeal, laryngeal, and facial nerves may follow, causing a **nasal quality** in the voice, difficulty in swallowing, and risk for aspiration.
- **Cranial neuropathies** characteristically occur in the 5th wk, leading to oculomotor and ciliary paralysis, which can cause strabismus, blurred vision, or difficulty with accommodation. Symmetric **polyneuropathy** has its onset 10 days to 3 mo after oropharyngeal infection and causes principally motor deficits with diminished deep tendon reflexes. Distal muscle weakness in the extremities with proximal progression is more commonly described than proximal muscle weakness with distal progression. Clinical and cerebrospinal fluid findings in the former are indistinguishable from those of Guillain-Barré syndrome. Paralysis of the diaphragm may ensue. Complete neurologic recovery is likely, but rarely, 2-3 wk after onset of illness, vasomotor center dysfunction can cause hypotension or cardiac failure.
- Recovery from the myocarditis and neuritis is often slow but usually complete. Corticosteroids do not diminish these complications and are not recommended

Treatment

- Specific antitoxin is the mainstay of therapy and should be administered on the basis of clinical diagnosis. Because it neutralizes only free toxin, antitoxin efficacy diminishes with elapsed time after the onset of mucocutaneous symptoms. Antitoxin is administered as a single empirical dose of 20,000-120,000 U based on the degree of toxicity, site and size of the membrane, and duration of illness.
- Suggested dose ranges are: pharyn-geal or laryngeal disease of 2 days' duration or less, 20 000 to 40 000 U; nasopharyngeal lesions, 40 000 to 60 000 U; extensive disease of 3 or more days' duration or diffuse swelling of the neck, 80 000 to 120 000 U. Antitoxin probably is of no value for cutane-ous disease, but some experts recommend 20 000 to 40 000 U of antitoxin, because toxic sequelae have been reported

- Antitoxin is probably of no value for local manifestations of cutaneous diphtheria, but its use is prudent because toxic sequelae can occur. Commercially available intravenous immunoglobulin preparations contain low titers of antibodies to diphtheria toxin; their use for therapy of diphtheria is not proven or approved. Antitoxin is not recommended for asymptomatic carriers.
- The role of antimicrobial therapy is to halt toxin production, treat localized infection, and prevent transmission of the organism to contacts. *C. diphtheriae* is usually susceptible to various agents in vitro, including penicillins, erythromycin, clindamycin, rifampin, and tetracycline. Resistance to erythromycin is common in populations if the drug has been used broadly.
- Antimicrobial Therapy. Erythromycin administered orally or parenterally for 14 days,penicillin G administered intramuscularly or intravenously for 14 days, or penicillinG procaine administered intramuscularly for 14 days constitute acceptable therapy.Antimicrobial therapy is required to stop toxin production, to eradicate C diphtheriae,and to prevent transmission but is not a substitute for antitoxin, which is the primary therapy. Elimination of the organism should be documented 24 hours after completion of treatment by 2 consecutive negative cultures from specimens taken 24 hours apart.
- Antibiotic therapy is not a substitute for antitoxin therapy. Some patients with cutaneous diphtheria have been treated for 7-10 days. Elimination of the organism should be documented by negative results of at least 2 successive cultures of specimens from the nose and throat (or skin) obtained 24 hr apart after completion of therapy. Treatment with erythromycin is repeated if either culture yields *C. diphtheria*
- Immunization. Active immunization against diphtheria should be undertaken during convalescence from diphtheria; disease does not necessarily confer immunity. Cutaneous Diphtheria. Thorough cleansing of the lesion with soap and water and administration of an appropriate antimicrobial agent for 10 days are recommended. Carriers. If not immunized, carriers should receive active immunization promptly, and measures should be taken to ensure completion of the immunization schedule. If a carrier has been immunized previously but has not received a booster of diphtheria toxoid within 5 years, a booster dose of a vaccine containing diphtheria toxoid (DTaP, Tdap, DT, or Td, depending on age) should be given. Carriers should be given oral erythromycin or penicillin G for 10 to 14 days or a single intramuscular dose of penicillin G benzathine (600 000 U for children weighing less than 30 kg and 1.2 million U for children weighing 30 kg or more and adults). Two follow-up cultures should be obtained after completing antimicrobial treatment to ensure detection of relapse, which occurs in as many as 20% of patients treated with erythromycin. The rst culture should be obtained 24 hours aftercompleting treatment. If results of cultures are positive, an additional 10-day course of
- ISOLATION OF THE HOSPITALIZED PATIENT: In addition to standard precautions, droplet precautions are recommended for patients and carriers with pharyngeal diphtheria until 2 cultures from both the nose and throat collected 24 hours after completing antimicro- bial treatment are negative for C diphtheriae. Contact precautions are recommended for patients with cutaneous diphtheria until 2 cultures of skin lesions taken at least 24 hours apart and 24 hours after cessation of antimicrobial therapy are negative