Fever and Sore Throat

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FIFTH YEAR

Learning Objectives

- Determine the etiology of fever and sore throat?
- Define the concept, causes, and clinical manifestations of diphtheria?
- Outline management of diphtheria?
- Define the concept ,causes ,and clinical manifestations of infectious mononucleosis (IMN)?
- Outline management and complications of infectious mononucleosis ?

Diphtheria

Diphtheria

- Greek *diphthera* (leather hide)
- Caused by Aerobic Gram +ve rods
- Cornyebacterium diphtheriae
- Exotoxin production only if infected by virus phage infected carrying toxin gene

Epidemiology

- ***Sources of infection***
  - **Patients and asymptomatic carriers**
  - **Patients: Transmission time is variable, usually persist 12 days or less, and seldom more than 4 weeks, without antibiotics.**

Epidemiology

- ***Susceptibility***
  - **The susceptibility are influenced by widespread immunization in childhood and immunity obtained after infection.**
  - **Children of 2-10 years old before widespread immunization.**
  - **the unimmunized or inadequately immunized adults after widespread immunization.**

Diphtheria Epidemiology

• Reservoir	Human carriers Usually asymptomatic
• Transmission	Respiratory Skin and fomites rarely
• Temporal pattern	Winter and spring
• Communicability	Up to several weeks without antibiotics

Etiology

- **There are three biotypes — gravis, intermedius, and mitis. The most severe clinical type of this disease is associated with the gravis biotype, but any strain may produce toxin.**

Pathogenesis and pathology

- **The organism produces a toxin that inhibits cellular protein synthesis and is responsible for local tissue destruction and pseudomembrane formation.**

Pathogenesis and pathology

- The pseudomembrane consists of coagulated fibrin, inflammatory cells, destructed mucous tissues and bacteria.
- the pseudomembrane in larynx, trachea or bronchia may have the potential for airway obstruction.

Pathogenesis and pathology

- **The toxin produced at the site of the pseudomembrane is absorbed into the bloodstream and then distributed to the tissues of the body.**

Pathogenesis and pathology

- **The toxin is responsible for the major complications of myocarditis and neuritis, and can also cause low platelet counts (thrombocytopenia) and protein in the urine (proteinuria).**

Pathogenesis and pathology

- **The rapidity of onset, the severity of disease, and the ultimate outcome are determined by the site of infection, the virulence of the strain and the status of host immunization, in actual, by the site and magnitude of the local lesions (pseudomembrane).**

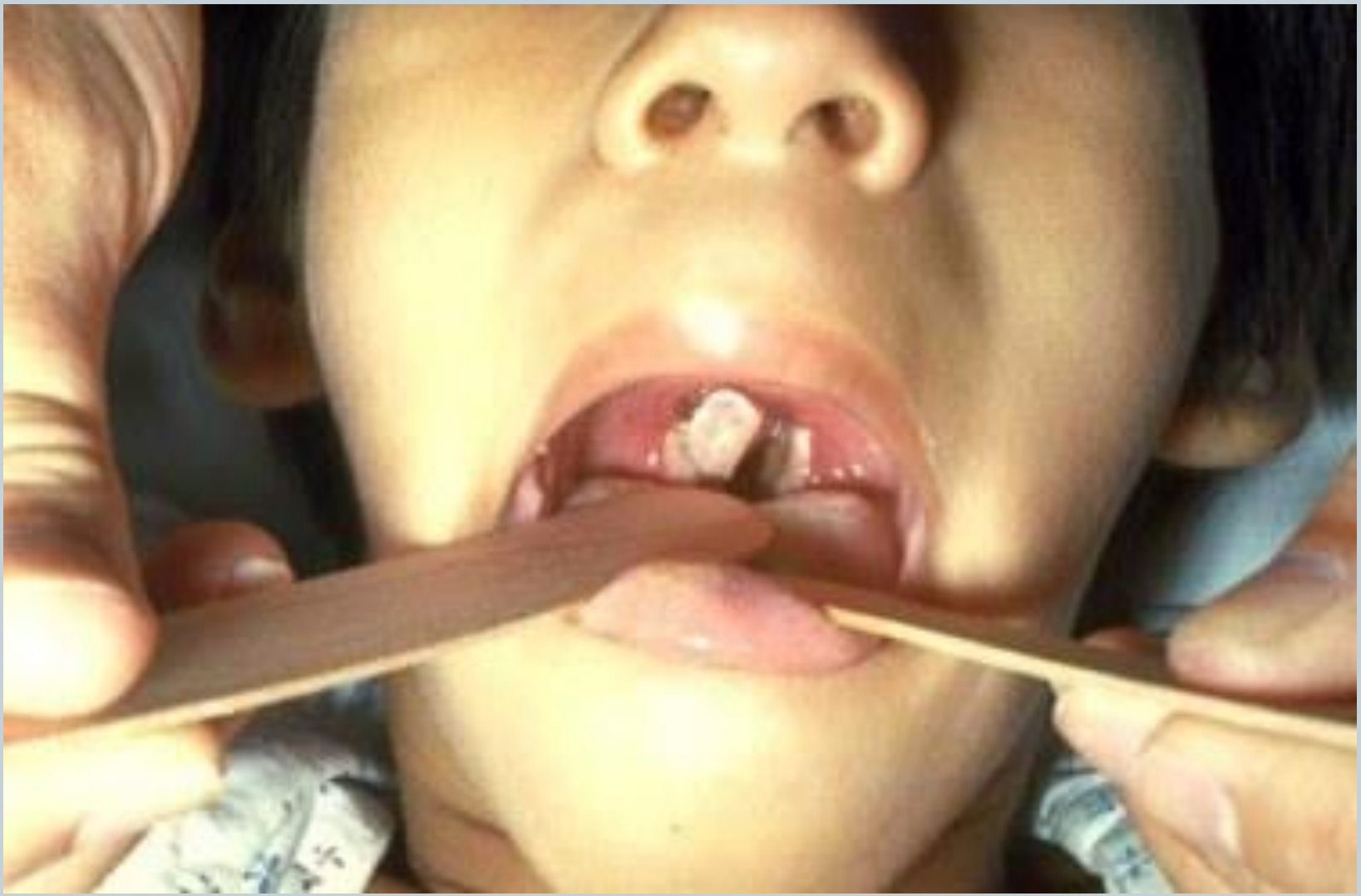
Clinical Manifestations

- Incubation period 2-5 days (range, 1-10 days)
- May involve any mucous membrane
- Classified based on site of infection
  - anterior nasal
  - pharyngeal and tonsillar
  - laryngeal
  - cutaneous
  - ocular
  - genital

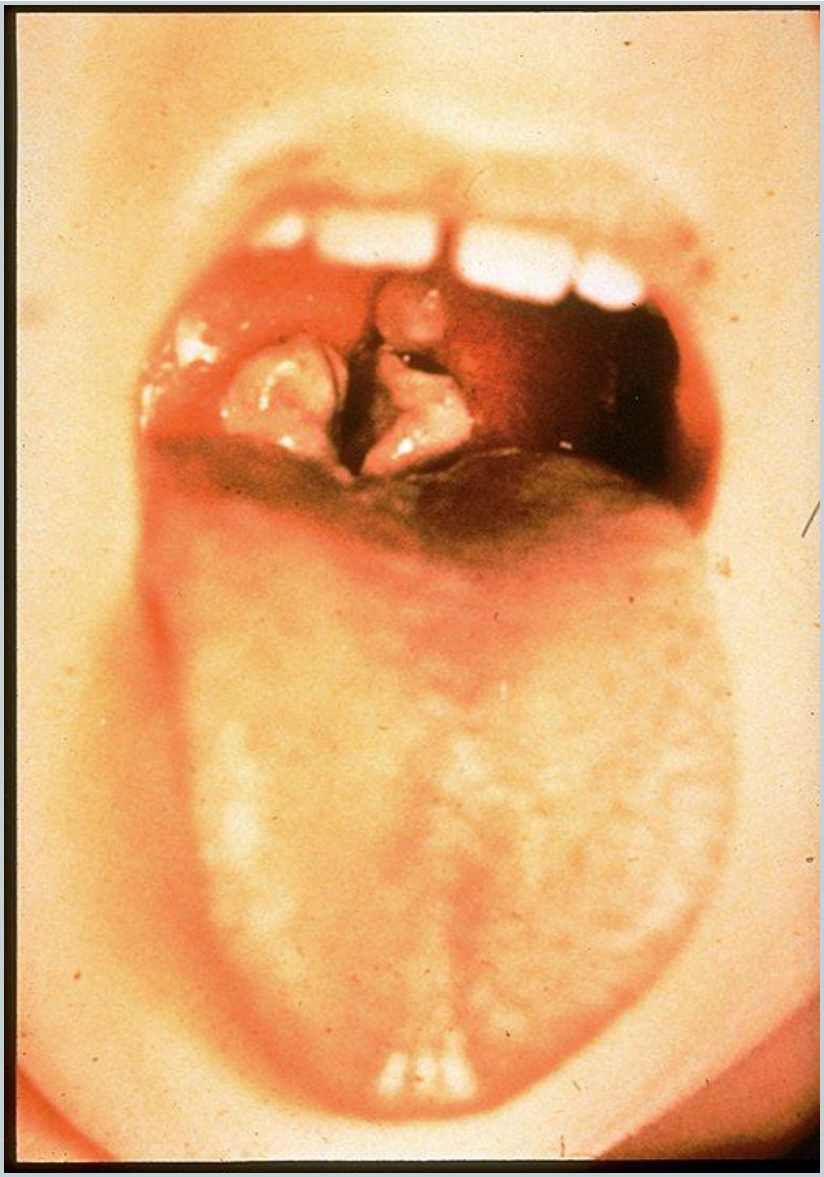
Pharyngeal and Tonsillar Diphtheria

- Insidious onset of exudative pharyngitis
- Exudate spreads within 2-3 days and may form adherent pseudo membrane
- Membrane may cause respiratory obstruction
- Fever usually not high but patient appears toxic

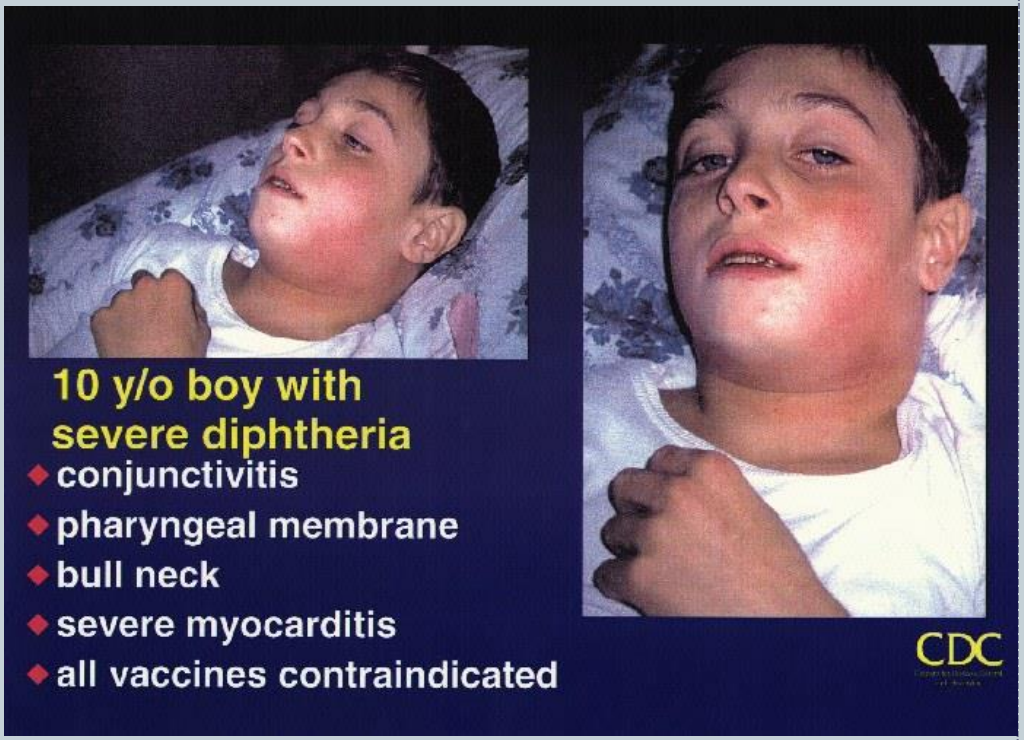
Thick Membrane

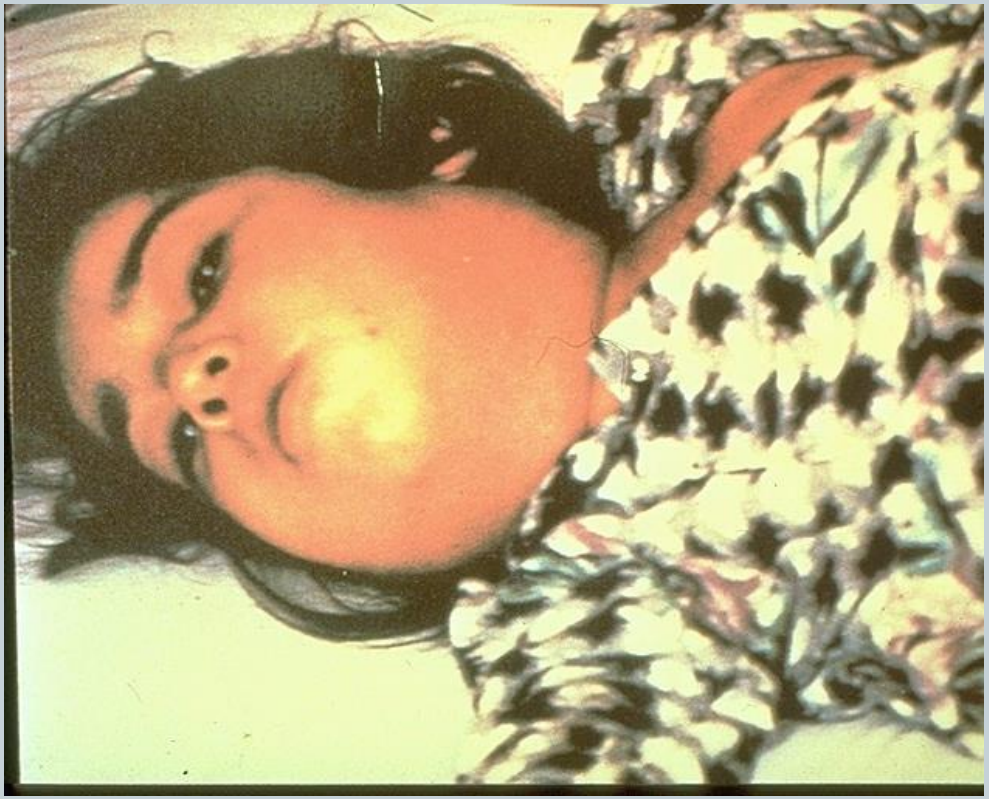


Pseudo membrane

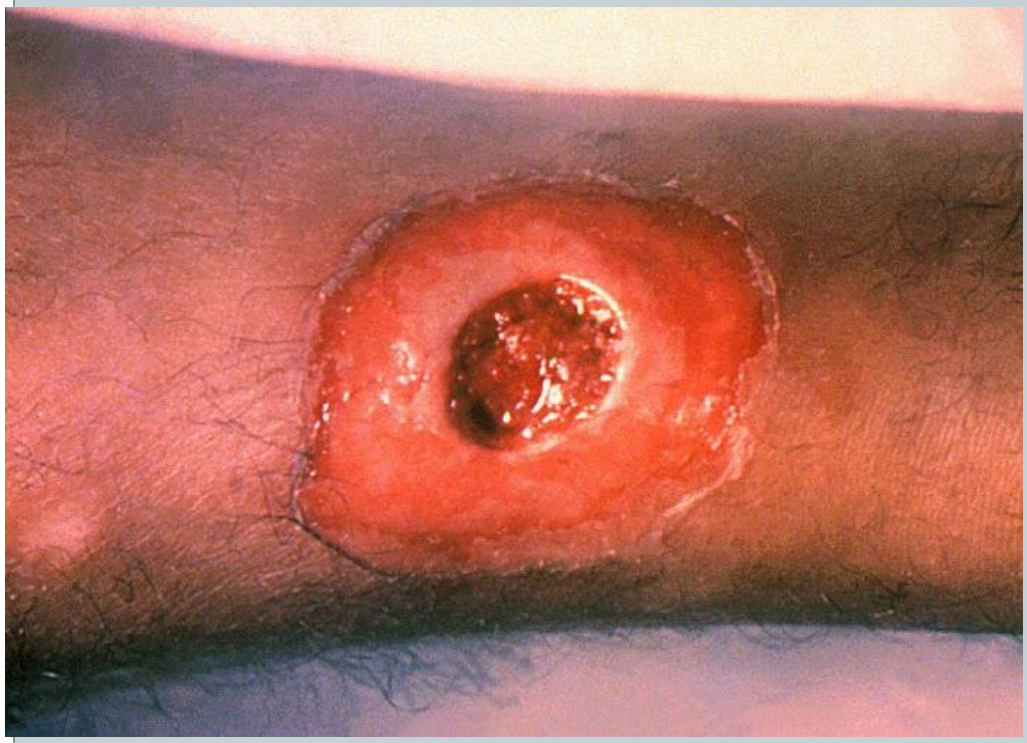


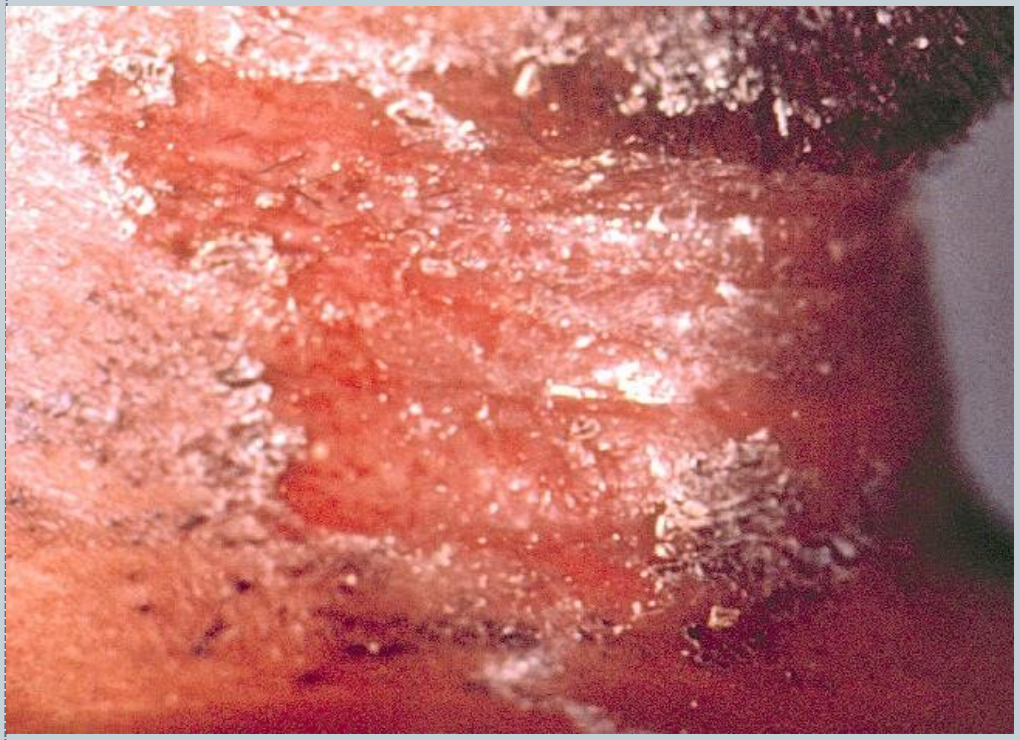
‘Bull Neck’


10 y/o boy with
severe diphtheria
conjunctivitis
pharyngeal membrane
bull neck
severe myocarditis
all vaccines contraindicated
CDC



Skin Lesions





Laboratory findings

- **Routine examination**
  - **Leukocytosis, 10~20 G/L, neutrophil is dominant.**
  - **Low platelet count (thrombocytopenia), rise profiles of the serum enzyme tests and proteinuria were found in serious cases.**

Laboratory findings

- **Bacteriological examinations**
  - **Smear and gram stain can found *C. diphtheriae*, but can not identify from the diphtheroids.**
  - **Fluorescent antibody-stain can found toxigenic *C. diphtheriae*, favourable for early diagnosis, but definitive diagnosis (false positive).**

Treatments

- **Strict isolation**
- **Use antitoxin and antibiotics for neutralization of free toxin, elimination of further toxin production and to control local infection.**
- **Use supportive interventions during disintoxication.**

Treatments

- ***General measures***
  - **Relax on bed for more than 3 weeks, 4-6 weeks for patients with myocarditis.**
  - **Provide adequate energy and nutriments**
  - **Fluorescent antibody-stain can found toxigenic *C. diphtheriae*, favourable for early diagnosis, but definitive diagnosis (false positive).**

Treatments

- ***Diphtheria antitoxin***
  - Diphtheria antitoxin, produced in horses.
  - It will not neutralize toxin that is already fixed to tissues, but will neutralize circulating toxin.
  - Early use will prevent progression of disease.
  - **The earlier, the better.**

Treatments

- ***Antibiotics***
  - **Procaine penicillin G daily, intramuscularly (300,000 U/day for those weighing 10 kg or less and 600,000 U/day for those weighing more than 10 kg) for 7-10 days.**
  - **Erythromycin orally or by injection (40-50 mg/kg/day; maximum, 2 gm/day) for 14 days.**

Diphtheria Complications

- Mostly attributable to toxin
- Severity generally related to extent of local disease
- Most common complications are myocarditis and toxic neuritis with palsy
- Death occurs in 5%-10% for respiratory disease

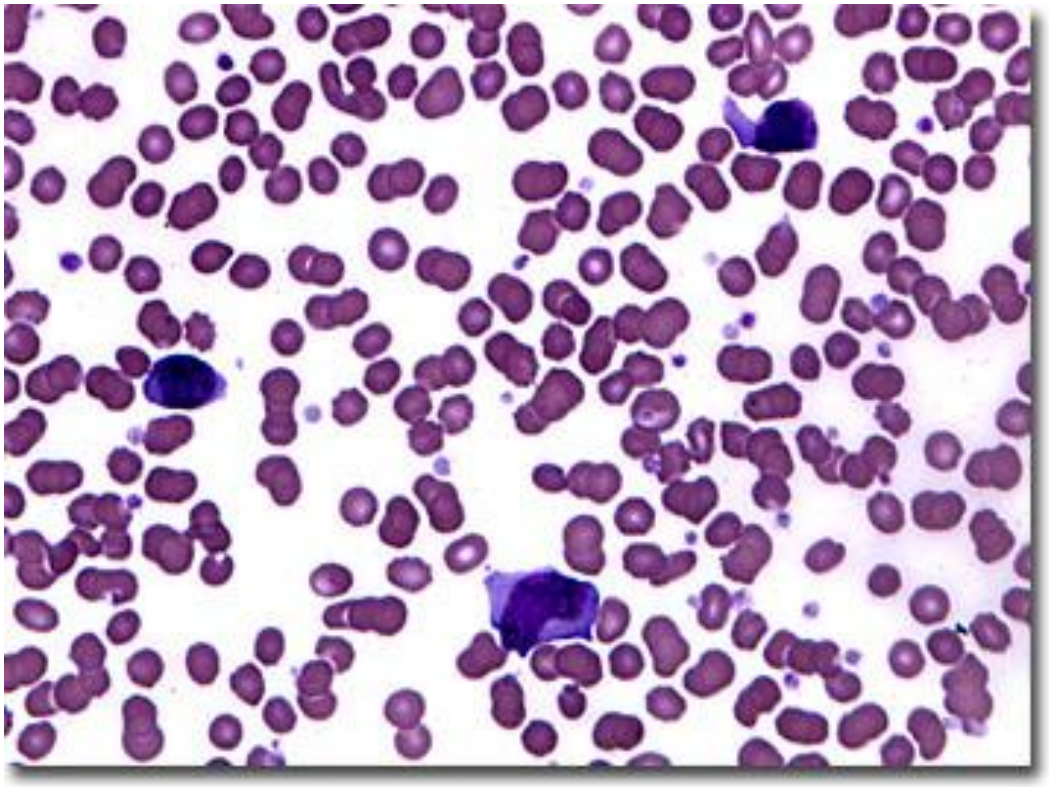
Preventions

- **Protect the susceptibles by vaccination**
  - **The effective measure**
  - **Primary series (DTP, multivalent vaccine) given at age of 3, 5, 6 months.**
  - **Boosters (DTP) given at 15 months and 4-6 years old, and booster (DT) every 10 years after then.**

Prognosis

- **The overall case-fatality rate for diphtheria is about 5%, with higher death rates (up to 20%) in persons <5 and >40 years of age.**

Infectious Mononucleosis



Virology

- Epstein Barr Virus (EBV)
  - Herpes Family – (linear DNA virus HHV4)
  - Surrounded by nucleocapsid and glycoprotein envelope
- Also associated w/ nasopharyngeal carcinoma, Burkitts lymphoma, Hodgkins Disease, B cell lymphoma.

Epidemiology

- Worldwide Prevalence of EBV
- Infections peak in early childhood and late adolescence/young adulthood.
- By adulthood , 90% of individuals have been infected and have antibodies to the virus.

Pathogenesis

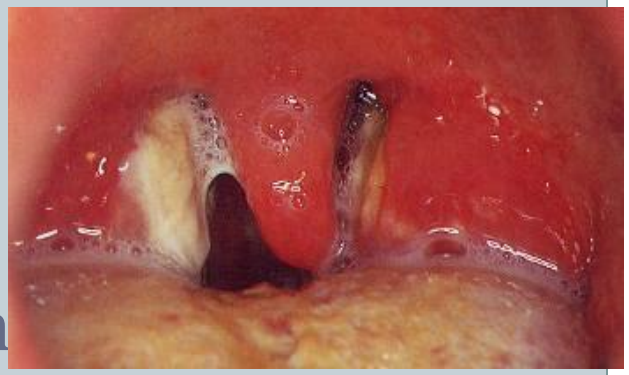
- EBV infects the epithelium of the oropharynx and salivary glands.
- Lymphocytes in the tonsilar crypts are directly infected -> BLOODSTREAM.
- Infected B cells and activated T cells proliferate and expand.
- Polyclonal B cells produce antibodies to host and viral proteins.

Infectious Mononucleosis Pathogenesis

- Memory B cells (not epithelial cells) are reservoir for EBV.
- EBV receptor is CD21 (found on B cell surface)
- Cellular immunity (suppressor T cells, NK cells, cytotoxic T cells) more important than humoral immunity in controlling infection

Signs & Symptoms

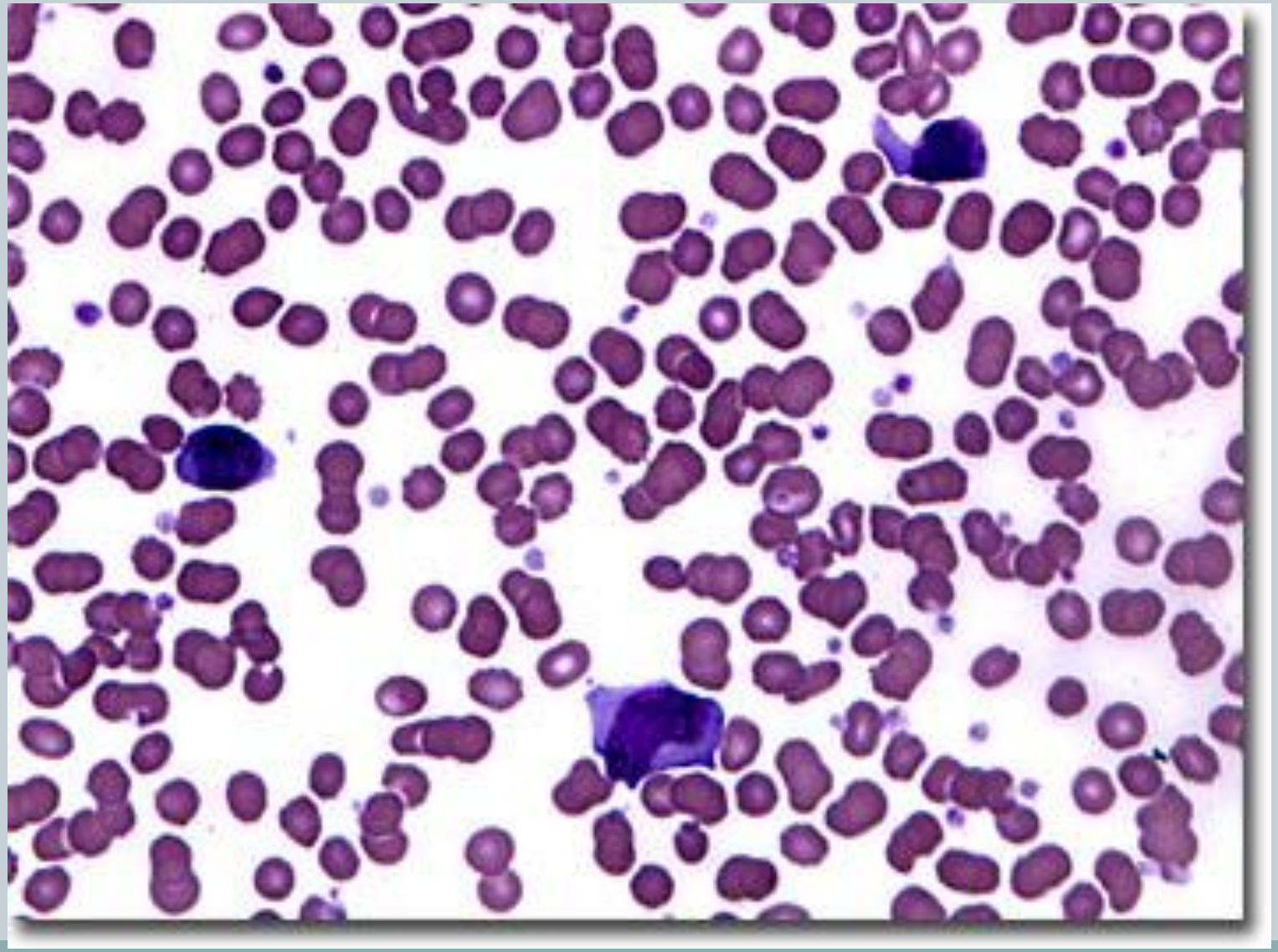
- Incubation 4-6 wks
- Prodrome (1-2 weeks before illness) Fatigue, Malaise, Myalgias
- Symptoms
  - Sore throat, Malaise, Headache, Abdom Nausea/Vomiting, Chills
- Signs
  - Lymphadenopathy, Fever, Pharyngitis, Splenomegay, Hepatomegaly, Rash, Periorbital Edema, Palatal Enanthem, Jaundice.



Diagnosis

- Lymphocytosis (>50% Lymphs)
- Atypical Lymphocytes (>10%, mostly CD8+ T cells)
- +Heterophile Antibodies (human serum agglutinates the erythrocytes of non-human species) (75% sens, 90% spec) (FP = lymphoma, CTD, viral hepatitis, malaria)
- Monospot -rapid agglutination assay – lower sens
- Confirm dx w/ antibodies to viral capsid antigen (VCA), early antigens (EA) and EBNA
- LFTs abnormal in 90%

Diagnosis

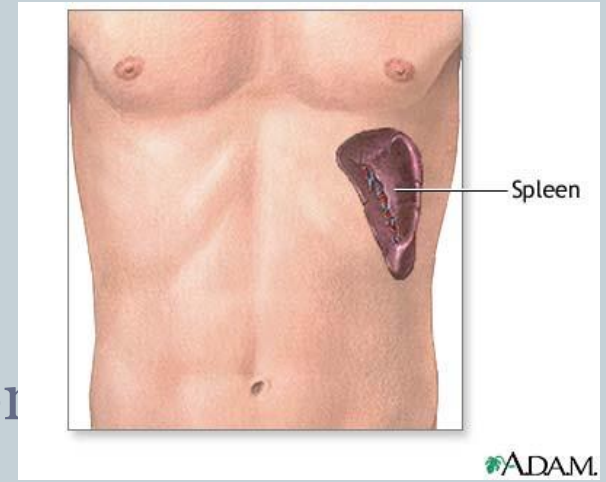


Treatment

- Rest
- Analgesics
- Avoid excessive physical activity (risk for splenic rupture).
- Prednisone for severe airway obstruction, hemolytic anemia, or thrombocytopenia.
- No role for acyclovir

Prognosis

- Most cases are self limited
- Complications include
  - Meningitis/Encephalitis (<1%)
  - Splenic rupture (0.1-0.2%)
  - Upper airway obstruction (<1%)
  - Bacterial superinfection
  - Autoimmune hemolytic anemia (3%) (Coo
    Agglutnins)



THANK YOU