

# DIPHTHERIA

## DEFINITION

Diphtheria is an acute infectious disease of childhood caused by *Corynebacterium diphtheriae* and clinically characterized by pseudomembrane formation at the site of infection and deleterious toxæmic effects on several tissues especially the myocardium and nervous tissue.

## ETIOLOGY

The causative agent *Corynebacterium diphtheriae* ( coryne = club & diphtheroes = leather ) is also known as Kleb-Löffler's bacilli. Some strains of *C. diphtheriae* produce diphtheria toxin, a protein that can cause myocarditis, polyneuritis, and other systemic toxic effects. Respiratory diphtheria is usually caused by toxinogenic *C. diphtheriae*, but cutaneous diphtheria is frequently caused by nontoxinogenic strains.

## MICROBIOLOGY

*C. diphtheriae*: aerobic, pleomorphic, nonmotile, nonsporulating, irregularly staining, gram<sup>+</sup> bacillus, club-shaped, arranged in clusters (Chinese letters/Cuneiform pattern) or parallel arrays (palisades) & forms gray to black colonies on selective media containing tellurite.

There are 3 biotypes viz. *gravis*, *mitis*, and *intermedius*

## EPIDEMIOLOGY

### Problem statement

In the tropics, cutaneous diphtheria is more common than respiratory diphtheria, occurs throughout the year, and often develops as a secondary infection complicating other dermatoses. Cutaneous diphtheria is increasingly recognized in temperate climates and accounted for most of the cases in the Seattle epidemic of 1972 to 1982.

### Agent factors

Source of infection : cases (subclinical/frank clinical) & carriers (temporary/chronic; nasal/throat)

Mode of infection : droplet infection

Infective material : nasopharyngeal secretion/ discharge of skin lesions/fomites/infected dust

Infective period : 2-4 weeks from the onset of disease.

### Host factors

Age : 1- 5 years (more common in children)

Sex : both

Immunity : Schick test surveys in India show that 70% of children below 3 yrs age & 90% of children below 5 yrs age are already immune to diphtheria.

Fatality rate : 10% in untreated and 5% in treated patients.

## Infections

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Humans are the principal reservoir for *C. diphtheriae*. Transmission occurs primarily by close personal contact. *C. diphtheriae* will be transmitted to susceptible individuals from patients with diphtheria than from carriers.

Case/carrier is considered non communicable when atleast two cultures properly obtained from nose & throat 24 hrs apart are negative for diphtheria bacilli.

Incubation period for respiratory diphtheria = 2 to 5 days and rarely up to 8 days.

Incubation period for cutaneous diphtheria = 7 days and rarely up to 21 days.

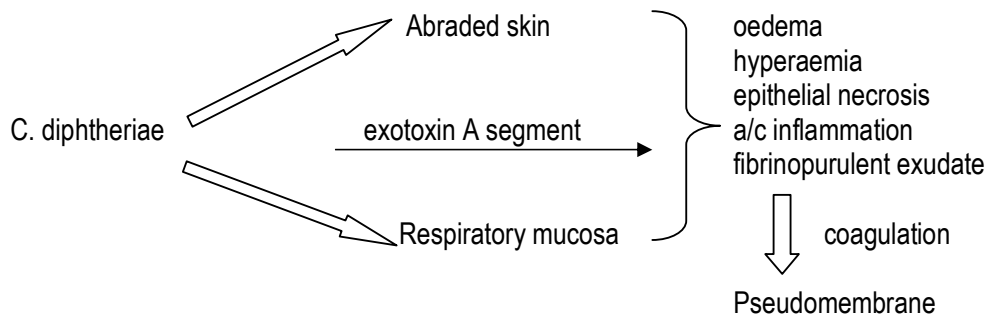
## Environmental factors

Especially the disease is seen in winter season

## PATHOLOGY AND PATHOGENESIS

Diphtheria exotoxin has 2 segments, A & B. Segment A is active & it acts both locally & systemically.

### Local effects



The pseudomembrane is adherent & when removed forcibly it leaves a raw bleeding surface. The pseudomembrane contains large numbers of *C. diphtheriae* organisms, but the bacteria are rarely isolated from the blood or internal organs.

### Systemic effects

Myocarditis, neuritis & focal necrosis in various organs, including the kidneys, liver, & adrenal glands.

Myocardial changes : cloudy swelling of muscle fibers and interstitial edema.

These changes are followed within weeks by hyaline and granular degeneration (sometimes with fatty degeneration), progressing to myolysis and finally to the replacement of lost muscle by fibrosis.

Thus, diphtheria can cause permanent cardiac damage.

In diphtheritic polyneuritis, pathologic changes include patchy breakdown of myelin sheaths in peripheral and autonomic nerves, but recovery of nerve damage is the rule if the patient survives.

## CLINICAL MANIFESTATIONS

- ❖ Onset is often gradual, but most patients seek medical care within a few days of illness.
- ❖ Fever of 37.8 °C to 38.9 °C , sore throat, and weakness -most common symptoms.

- ❖ Dysphagia, headache, and change of voice occur in fewer than half of patients.
- ❖ Neck edema and dyspnoea are noted in some patients which may be fatal.
- ❖ Patients without toxicity exhibit discomfort and malaise associated with local infection.
- ❖ Severely toxic patients may develop listlessness, pallor, and tachycardia that can progress rapidly to vascular collapse.

### CLINICAL TYPES

**Nasal diphtheria** : occurs in 2-3% cases.

- ❖ Pseudomembrane limited to nasal septum & turbinates of one side
- ❖ Foul smelling serosanguinous discharge
- ❖ Frank epistaxis

**Tonsillopharyngeal** : commonest type

Isolated spots of gray or white exudate may appear first. These spots often extend and coalesce within a day to form a confluent, sharply demarcated pseudomembrane that becomes progressively thicker, more tightly adherent to the underlying tissue, and darker gray in color. Dislodging the membrane is likely to cause bleeding.

A small percentage of patients present with **malignant or "bull-neck" diphtheria**, with extensive pseudomembrane formation, foul breath, massive swelling of the tonsils and uvula, thick speech, cervical lymphadenopathy, striking edematous swelling of the submandibular region and anterior neck, and severe toxicity.

**Laryngeal diphtheria** : 25% Cases

- ❖ hoarseness
- ❖ barking cough
- ❖ dyspnoea, stridor & cyanosis
- ❖ infants refuse to suck the breast due to choking

**Cutaneous diphtheria** ( *C.diphtheriae* can't penetrate intact skin.)

- ❖ Occurs in pre existing dermatoses involving the upper & lower extremities, head or trunk.
- ❖ The clinical features are similar to those of other secondary cutaneous bacterial infections.
- ❖ In the tropics, cutaneous diphtheria may present as a primary cutaneous lesion, typically with morphologically distinct "punched-out" ulcers that are covered by necrotic slough or membrane and have well-demarcated edges.

*C. diphtheriae* is an occasional cause of invasive infections, including endocarditis and septic arthritis. Risk factors for such infections include preexisting cardiac abnormalities, abuse of intravenous drugs, and alcoholic cirrhosis. Primary or secondary diphtheritic infection occasionally involves other mucous membranes, including the conjunctiva and the membranes of the genitourinary and gastrointestinal tracts.

### **COMPLICATIONS**

- **Obstruction of the respiratory tract** can be caused by extensive pseudomembrane formation and swelling early in the disease or by sloughed pseudomembrane that

becomes lodged in the airways later in the disease. The risk is greater when infection involves the larynx or the tracheobronchial tree and in children because of the small size of the airways.

- **Myocarditis and polyneuritis** are the most prominent toxic manifestations of diphtheria.
- **Post diphtheritic paralysis** :Bulbar dysfunction in diphtheritic neuritis typically develops during the first 2 weeks. Palatal and pharyngeal paralysis usually develops first. Swallowing is difficult, the voice is nasal, and ingested fluids may be regurgitated through the nose. Additional bulbar signs may develop over several weeks, with oculomotor and ciliary paralysis more common than facial or laryngeal paralysis. Peripheral polyneuritis typically begins from 1 to 3 months after the onset of diphtheria with proximal weakness of the extremities, which spreads distally. Paresthesia may occur, most often in a glove-and-stocking distribution. Polyneuritis usually resolves completely, with the time needed for improvement approximately equal to that elapsing from exposure to the development of symptoms.
- **Pneumonia** occurs in more than one-half of fatal cases of diphtheria.
- Less common complications include renal failure, encephalitis, cerebral infarction, pulmonary embolism, and bacteremia or endocarditis due to invasive infection by *C. diphtheriae*.
- Serum sickness may result from antitoxin therapy.

## DIAGNOSIS

**Clinical picture** :A characteristic pseudomembrane on the mucosa of the tonsils, palate, oropharynx, nasopharynx, nose, or larynx suggests diphtheria but is not uniformly present. Diphtheria should be considered in patients with sore throat, cervical adenopathy or swelling, and low-grade fever, especially when these manifestations are accompanied by systemic toxicity, hoarseness, stridor, palatal paralysis, or serosanguineous nasal discharge with or without demonstrable pseudomembrane. Cutaneous diphtheria may present as a characteristic "punched-out" ulcer with a membrane, but it is more often indistinguishable from other inflammatory dermatoses.

**Isolation of *C. diphtheriae* from local lesions** helps definitive diagnosis of diphtheria. The laboratory should be notified that diphtheria is suspected to ensure the use of selective tellurite medium appropriate for the isolation of *C. diphtheriae*. All isolates of *C. diphtheriae* should be subjected to toxicity testing.

Patients with *C. diphtheriae* in the respiratory tract are classified as diphtheria cases if pseudomembranes are present and as diphtheria carriers if pseudomembranes are absent. The disease is graded as tonsillar if pseudomembranes are localized to the tonsils, as combined types or delayed diagnosis if more extensive pseudomembranes are present, and as severe if cervical adenopathy or cervical edema is also present.

## Schick test

It is an intradermal test mostly used for immunological survey rather than diagnosis. It tests 2 things

- a) Presence of antitoxin indicating immunity status

b) State of hypersensitivity to diphtheria toxin / other proteins of diphtheria cells

Method : Intradermal injection of

- 0.2 ml of schick test toxin into the skin of forearm ( test arm ) &
- 0.2 ml of same toxin inactivated by heat / 0.1 ml of fluid toxoid vaccine preparation to opposite arm ( control arm )

Reactions seen	Test arm	Control arm	Inference
Negative immune.	No reaction	No reaction	Person is
Positive Susceptible	Typical red flush*	No reaction	
Pseudopositive reaction.	Smaller flush	Smaller flush	Allergic
Combined Susceptible	Typical red flush*	Smaller flush	

\* Typical red flush denotes a circumscribed red flush of 1-5 cm diameter occurring 24 – 36 hours of reaction & reaching its maximum development by 4<sup>th</sup> – 7<sup>th</sup> day.

**DIFFERENTIAL DIAGNOSIS**

D/D of nasal diphtheria : foreign body in nose,rhinorrhoea, snuffles due to congenital syphilis

D/D of tonsilopharyngeal diphtheria

- a/c streptococcal membranous tonsillitis (high temp.;memb. Confined to tonsils)
- viral membranous tonsillitis ( total count of WBC normal / low )
- herpetic tonsillitis ( extremely painful lesions on tongue & palate)
- oral thrush ( moniliasis; no systemic symptoms; thrush membrane in tongue & buccal mm)
- IMN ( with abnormal lymphocytes in blood; Paul Bunnell test is confirmatory )
- Agranulocytosis & leukaemia ( with anaemia & haemorrhagic lesions )
- Post tonsillectomy faucial membrane ( without spread )

D/D of laryngeal diphtheria : croup, epiglottitis, retropharyngeal abscess, peritonsillar abscess

**ALLOPATHIC MANAGEMENT**

Diphtheria antitoxin ,Epinephrine ,Erythromycin, penicillin G, rifampin, or clindamycin & DPT vaccine.

**HOMOEOPATHIC MANAGEMENT**

Arum, Brom, Crot-h, Diph, Kali-bi, Lac-c, Lyc, Merc-b-i, Merc-cyan, Merc-p-l, Phyt, Sabad (H.C Allen)

Simple type : Bell, Phyt

Croupal : Apis, Canth, Merc-sol, Merc-b-l,Merc-p-l,Brom, Kali-bi

Malignant/septic : Mur-ac,Lach, Kali-mang, Calc-chlor,Carb-ac

Post diphtheritic paralysis : Gels, Cocculus

## MUMPS

### DEFINITION

Mumps, caused by Myxovirus parotiditis which has a predilection towards glandular & nervous tissue, is an acute, systemic, communicable infection whose most distinctive feature is non suppurative enlargement & tenderness of one or both parotid glands. Involvement of other salivary glands, the meninges, the pancreas, and the gonads is also common.

### ETIOLOGY

Myxovirus parotiditis, a paramyxovirus, is pleomorphic and has a diameter ranging from 100 to 600 nm. The virion is composed of RNA and five proteins. The RNA is surrounded by an envelope with glycoprotein projections. There are two envelope glycoproteins a haemagglutinin-neuraminidase (HN) and a haemolysis cell fusion antigen (F) as well as a matrix envelope protein (M). There are two internal components: a nucleocapsid protein (NP) and an RNA polymerase protein. There is only one antigenic type of mumps virus.

### EPIDEMIOLOGY

Distribution	: Worldwide
Occurance	: endemic
Secondary Attack Rate	: 86 %
Spread	: Man ↔ man
Immunity	: lifelong ( but second attacks can occur )

### PATHOGENESIS

Mumps virus is transmitted by droplet nuclei, saliva, and fomites. Virus is present in saliva for 7 days before & 8 days after onset of parotitis. Replication of the virus in the epithelium of the upper respiratory tract leads to viremia, which is followed by infection of glandular tissues and/or the central nervous system (CNS).The affected glands contain perivascular and interstitial mononuclear cell infiltrates with prominent edema. Necrosis of acinar and epithelial duct cells is evident in the salivary glands and in the germinal epithelium of the seminiferous tubules.

Parotitis, if it develops, usually does so within the next 24 h but may be delayed for as long as a week; it is generally bilateral, although the onset on the two sides may not be synchronous and at times only one side is affected. The submaxillary and sublingual glands are involved less often than the parotid and are almost never involved alone.

### CLINICAL MANIFESTATIONS

- ❖ Fever, malaise, myalgia, HA & anorexia ( prodromal symptoms )
- ❖ Tenderness and obliteration of the space bet. the ear lobe and angle of the mandible.
- ❖ The patient frequently reports an earache and finds it difficult to eat, swallow, or talk.

- ❖ Trismus (lock jaw) and dryness of mouth (xerostomia)
- ❖ Glandular swelling increases for a few days and then gradually subsides, disappearing within a week. The orifice of Stensen's duct is commonly red and swollen.
- ❖ Redness & oedema may not be seen ( seen only in suppurative parotitis).
- ❖ Presternal pitting edema has been described in about 5% of mumps cases, often in association with submandibular adenitis.

IP : 12 – 21 days

Subclinical course : 30 – 40 %

Fever & glandular enlargement subsides after 5 – 7 days.

### COMPLICATIONS

- Orchitis is the most common manifestation of mumps among postpubertal males, developing in about 20% of cases. The testis is painful and tender and is enlarged to several times its normal size; accompanying fever and abdominal pain is common. Later, testicular atrophy develops in half of the affected men. Since orchitis is bilateral in fewer than 15% of cases, **sterility after mumps is rare.**
- Oophoritis in women is far less common than orchitis.
- Aseptic meningitis (Symptoms include stiff neck, headache, and drowsiness)
- Mumps pancreatitis, may present as abdominal pain,
- An excessive number of spontaneous abortions are associated with gestational mumps when the disease occurs during the first trimester. Mumps in pregnancy does not lead to premature birth or fetal malformations.
- Rare complications : Nerve deafness, Polyarthritis, cerebellar ataxia, facial palsy, transverse myelitis, Guillain-Barre syndrome, reactive hydrocoele and hydrocephalus,
- Myocarditis, mastitis, thyroiditis, nephritis, arthritis, and thrombocytopenic purpura.

### DIAGNOSIS

The diagnosis of mumps is made easily in patients with acute bilateral parotitis and a history of recent exposure. When parotitis is unilateral or absent or when sites other than the parotid gland are involved, laboratory diagnosis is required

- ✓ Leucopenia with relative lymphocytosis
- ✓ CSF pleocytosis in about 50% cases
- ✓ Isolation of virus after inoculation of appropriate clinical specimens into a variety of host systems, such as rhesus monkey kidney cells and human embryonic lung fibroblasts.
- ✓ Fluorescein-labeled monoclonal antibody tests
- ✓ Demonstration of virus in specimens from saliva, throat, and urine during the first few days of illness and from the CSF of patients with mumps meningitis
- ✓ Highly sensitive enzyme-linked immunosorbent assays
- ✓ Increase in IgG antibody titer or by the demonstration of specific IgM in one serum specimen.

### DIFFERENTIAL DIAGNOSIS

- Infection with parainfluenza virus type 3, coxsackieviruses, and influenza A virus
- Metabolic diseases, such as diabetes mellitus and uremia
- Drugs, such as phenylbutazone and thiouracil.
- Unilateral parotid swelling can result from a tumor, a cyst, or a ductal obstruction due to stones or strictures.
- Diseases with chronic parotid swelling like sarcoidosis, Sjogren's syndrome & AIDS.

- Suppurative parotitis, usually caused by *Staphylococcus aureus*, is most often unilateral and presents with redness & oedema which are absent in mumps
- Testicular torsion may produce a painful scrotal mass resembling that seen in mumps orchitis. Other viruses (e.g., enteroviruses) may cause aseptic meningitis that is clinically indistinguishable from that due to mumps virus.

## ALLOPATHIC MANAGEMENT

**Curative** :Therapy for parotitis and other manifestations of **mumps** is symptom-based along with the administration of analgesics.

**Prophylactic**:Measles-mumps-rubella (MMR) vaccine.  
Live attenuated mumps vaccine (Jeryl Lynn strain)

## HOMOEOPATHIC MANAGEMENT

### General

Warm or cold compresses to the affected side  
Soft diet with plenty of fluids  
Bed rest for 1-2 weeks

### Medicinal

#### Curative

Merc-sol, Puls, Lyssin ( Allen's keynotes )  
Acon, Merc-sol, Puls ( Richard Hughes)  
Ailanthus, Fagopyrum, Trifolium pretense, Trifolium repens ( Clarke's repertory)  
Acon, Bell, Brom, Merc-c, Merc-sol, Phyt, Puls, Rhus-t, Sil ( Boericke's repertory)

#### Indications

**Merc-sol** :Mumps, diphtheria, tonsillitis with profuse offensive saliva; tongue large, flabby with imprint of teeth; mapped tongue (Lach., Nat., Tarax.).

**Pulsatilla** :Mumps; metastasis to mammae or testicle.

**Lyssin** :Sensation as if she would have mumps.

**Bromium** :Left-sided mumps.

**Phytolacca** : Mumps. Follicular pharyngitis.

**Jaborandi (Pilocarpus)**: Dr. Burnett's homoeopathic remedy for Mumps seems to surpass all the rest. It acts very quickly, and also relieves the pain. Moreover, Pilocarpus has a reputation for the metastases in which Mumps excels, whether to testes or mammae; when the swelling suddenly subsides, as the result of a chill, and worse troubles supervene. Pilocarpus also acts as a prophylactic.

#### Prophylactic

**Parotidinum** has been used as a prophylactic against infection by mumps. The well-known complications which sometimes occur with mumps, cerebral inflammation and orchitis suggest its possible use in these conditions.

**Trifolium pretense** : ( White clover) Feeling as if mumps were coming on. (Prophylactic against mumps, feeling of congestion in salivary glands, pain and hardening, especially submaxillary; worse, lying down. Mouth filled with watery saliva, worse lying down. Taste of blood in mouth and throat. Sensation as if heart would stop, with great fear, better sitting up or moving about; worse, when alone, with cold sweat on face).