STAPHYLOCOCCAL & STREPTOCOCCAL INFECTIONS

	Staphylococcal infection	Streptococcal infections
Definition	an infection caused by pathogenic bacteria of one of several species of the genus *Staphylococcus* or their toxins. Almost any organ of the body may be involved. The infections occur in many forms, including meningitis, pneumonia, tonsillitis, urinary tract infection	an infection caused by pathogenic bacteria of one of several species of the genus *Streptococcus* or their toxins. Almost any organ of the body may be involved. The infections occur in many forms, including cellulitis, endocarditis, erysipelas, impetigo, meningitis, pneumonia, scarlet fever, tonsillitis, and urinary tract infection.
Etiology	Family - Micrococcaceae Genus -Staphylococcus Species: S. aureus, S. epidermidis, S. saprophyticus.	Family - Streptococcaceae Genus -Streptococcus Species: Str. pyogenes, Str. pneumoniae
Morphology	Gram-positive Spherical cell shape Nonmotile (non flagellated) Non spore forming non capsulated (rare strains are capsulated) Arranged in grape-like clusters (*staphyle* means bunch of grapes and this arrangement is due to the tendency of the organism to divide in different planes)	Gram-positive Spherical cell shape Nonmotile (non flagellated) Non spore forming capsulated or non capsulated Arranged in pairs or in chains (chain formation is due to the cocci dividing in one plane only and the daughter cells failing to separate completely).
Epidemiology	Source of infection: sick people, and staph carriers. Mode of transmission: direct or indirect contact with a person who has a discharging would, a clinical infection of the respiratory or urinary tract, or one who is colonised with the organism. Airborne transmission Fecal-oral transmission	Source of infection: sick people, and Streptococcus carriers Mode of transmission: direct or indirect contact with a person who has a discharging would, a clinical infection of the respiratory or urinary tract, or one who is colonised with the organism. Airborne transmission
Virulence factors	hyaluronidase, protease, coagulase, lipases, deoxyribonucleases and enterotoxins..	exotoxins, such as **pyrogenic (erythrogenic) toxin** which causes the rash of **scarlet fever** and systemic **toxic shock syndrome** **hyaluronidase**, and **streptolysins** **invasins** such as **streptokinase**, **streptodornase** (DNase B) **M-protein** to inhibit phagocytosis (3), *Streptococcus pyogenes* produces a wide array of **virulence factors** and a very large number of diseases. Virulence factors

		of Group A streptococci include: (1) **M protein**, fibronectin-binding protein (**Protein F**) and **lipoteichoic acid** for adherence; (2) **hyaluronic acid capsule** as an immunological disguise and to inhibit phagocytosis;
Methods of laboratory diagnostics	*Specimens :* depend on the site of the lesion e.g. Pus, blood, urine .sputum, faces, food remains and vomit from cases of food poisoning 1. Bacterioscopy - direct Gram stained smear 2. Bacteriological method: • isolation on culture media • Identification of the organism (catalase : positive; Coagulase: positive; DNAse : Positive; Mannitol fermentation: Positive) 3. Antibiotic sensitivity test	*Specimens :* depend on the site of the lesion e.g. Pus, blood, urine .sputum, faces, food remains and vomit from cases of food poisoning 1. Bacterioscopy - direct Gram stained smear 2. Bacteriological method: • isolation on culture media • Identification of the organism 3. Antibiotic sensitivity test
Treatment	-According to AB sensitivity test result -Penicillin -Cloxacillin -Aminoglucosides -Clindamycin -Vancomycin	-According to AB sensitivity test result -Penicillin Erythromycin or another macrolid
Specific prophylaxis		

Pathogenic Neisseriae: Gonorrhea, and Meningococcal Meningitis

	GONORRHEA	MENINGOCOCCAL MENINGITIS
Definition	Gonorrhoea is a highly contagious sexually transmitted disease that is caused by the bacterium Neisseria gonorrhoea.	Meningococcal meningitis is **a**n acute infectious caused by the bacteria *Neisseria meningitidis*
Etiology	Family – Neisseriaceae Genus – Neisseria	
	N. gonorrhoe	N. meningitidis
Morphology	Gram-negative diplococcus Nonmotile (non flagellated) Non spore forming	
	non capsulated	capsulated or non capsulated
Epidemiology	Source of infection: sick people. Mode of transmission: direct or indirect contact with a person who has a discharging would, a clinical infection of the respiratory or urinary tract, or one who is colonised with the organism.	Source of infection: sick people or Neisseria Meningitidis carrier Transmission: direct: droplet (coughing, sneezing)
Virulence factors	endotoxin	
Methods of laboratory diagnostics	Specimens for microscopic examination are obtained from the discharge of the urethra, vagina, vulva, cervix uteri, prostate, and conjunctiva. 1. Bacterioscopy - direct Gram stained smear 2. Bacteriological method: • isolation on culture media (ascitic agar, ascitic broth, and egg-yolk medium) • Identification of the organism 3. Serological tests (ELISA) 4. Antibiotic sensitivity test	Specimens: Cerebrospinal fluid, blood 1. Bacterioscopy - direct Gram stained smear 2. Bacteriological method: • isolation on culture media (blood agar • Identification of the organism 3. Serological tests (ELISA) 4. Antibiotic sensitivity test
Treatment	Antimicrobial therapy**:** penicillin G, ampicillin, ceftriaxone or ofloxacin	
Specific prophylaxis	There is no effective vaccine to prevent gonorrhoea	Meningococcal polysaccharide vaccine Meningococcal conjugate vaccine

Enteric Bacterial Infections

		ESCHERICHIOSIS	SHIGELLOSIS	TYPHOID FEVER PARATYPHOID FEVER A AND B	SALMONELLOSIS (FOOD POISONING)
Definition					
Etiology	Family	Enterobacteriaceae			
	Genus	Escherichia	Shigella	Salmonella	
	Species:	Escherichia coli	Sh.dysenteriae, Sh.flexneri Sh.boydii, Sh.sonnei	S. typhi, S. paratyphi A S.schottmuelleri	S.heidelberg, S. typhimurium S.derby
Morphology		Gram-negative Rod-shaped bacterium Non spore forming			
		Motile Capsulated or non capsulated	Nonmotile (non flagellated)	Motile	Motile
Epidemiology		Source of infection: sick people, carriers. Mode of transmission: Fecal-oral transmission is the main path of enteric infections. Other modes of transmission include ingestion of contaminated food or water, contact with infected objects.			Transmission to humans occurs via contaminated drinking water, milk, meat, and foods such as cake mixes that use contaminated ingredients; poultry and eggs
Virulence factors		Endotoxin; the capacity for adhesion and invasion; Hyaluronidase; Fibrinolysin; Letsitinaza; Hemolysins			
		Exotoxin	Exotoxin (Sh.dysenteriae)	do not produce exotoxin. -	do not produce exotoxin. -
Methods of laboratory diagnostics		Clinical specimens: blood, stool Bacteriological method: • isolation on culture media: Endo Agar (lactose-fermenting organisms such as E. coli produce characteristic red colonies. lactose non-fermenters form colorless, transparent colonies); Bismuth Sulfite Agar -WILSON-BLAIR (Salmonella typhi - black colonies; Salmonella spp. - black or greenish-gray colonies) • Identification: based on morphological, and biochemical properties (Hiss' media); Antigenic structure (Agglutination reaction). Serological tests: Widal test is an agglutination test for the detection of antibodies; ELISA; Immunofluorescence reaction. Antibiotic sensitivity test			

Treatment	Antimicrobial therapy**:** antibiotics (ciprofloxacin, tetracycline, chloramphenicol, ampicillin), sulfonamide antibiotic combination of trimethoprim and sulfamethoxazole, nitrofurans. Treatment of dehydration and hypotension (low blood pressure) is lifesaving in severe cases. Eubiotics: bifikol, bifidumbacterin.			
Specific prophylaxis	There is no effective vaccine to prevent Escherichiosis	There is no effective vaccine to prevent Shigellosis	There are two vaccines licensed for use for the prevention of typhoid: 1. the live, oral Ty21a vaccine 2. the injectable Typhoid polysaccharide vaccine	There is no effective vaccine to prevent Salmonellosis

Cholera

Definition	Cholera is an acute, diarrheal illness caused by infection of the intestine with the bacterium Vibrio cholerae.
Etiology	Family - Vibrionaceae Genus - Vibrio Species: - Vibrio cholerae, biovar El Tor&biovar Cholerae
Morphology	Gram-negative Comma-shaped bacterium Motile Non capsulated Non spore forming
Epidemiology	Source of infection: sick people, carriers. V. cholerae is usually transmitted via the ingestion of food or water contaminated (directly or indirectly) with feces or vomitus of infected persons. Mode of transmission: Cholera is transmitted by the fecal-oral route. Ingestion of contaminated food or water, contact with infected objects.
Virulence factors	Endotoxin and Exotoxin (Choleragen) Hyaluronidase; fibrinolysin; letsitinaza; hemolysins, coagulase, collagenase.
Methods of laboratory diagnostics	**Bacterioscopy** - direct Gram stained smear and hanging-drop test. **Bacteriological method**: • isolation on culture media: alkaline peptone water and alkaline agar • Identification: based on morphological, and biochemical properties; Antigenic structure (Agglutination reaction). **Serological tests**: Agglutination reaction, ELISA, Immunofluorescence reaction
Treatment	Oral or intravenous rehydration therapy (fluid replacement) Antimicrobial therapy: Tetracycline, Chloramphenicol.
Specific prophylaxis	Specific prophylaxis of cholera is performed by corpuscular vaccine and cholerogen-anatoxin

Plague

Definition	Plague is an acute, severe and potentially deadly bacterial infection usually transmitted through a flea bite and most commonly presents as bubonic, pneumonic or septicaemic forms. Plague is caused by the enterobacteria Yersinia pestis.
Etiology	Family – Enterobacteriaceae Genus – Yersinia Species - Yersinia pestis
Morphology	Gram-negative Capsulated Bipolar staining Nonmotile Rod-shaped bacterium Non spore forming.
Epidemiology	Source of infection: infected animal, usually a wild rodent (mice, rats, squirrels) or person who has plague infection in the lungs. Mode of transmission: • From animal to animal and from animal to human by the bite of an infected flea. • direct contact with the body fluids or tissues of a sick or dead animal • ingestion of raw or undercooked meat from an infected animal (marmots or prairie dogs, goat and camel) • airborne transmission (respiratory droplets from people or domestic pets with plague pharyngitis or pneumonia may transmit plague)
Virulence factors	Endotoxin and Exotoxin Hemolysin, fibrinolysin, hyaluronidase, coagulase.
Clinical forms	Bubonic, pneumonic or septicaemic forms.
Methods of laboratory diagnostics	Bacterioscopy - direct Gram stained smear (ovoid gram-negative organisms in material aspirated from buboes, sputum or CSF is highly suggestive of plague infection). Bacteriological method: • isolation on culture media • Identification: based on morphological, and biochemical properties; Antigenic structure (Agglutination reaction). Serological tests: Agglutination reaction, ELISA, Immunofluorescence reaction
Treatment	Antibiotics such as streptomycin, gentamicin, doxycycline, or ciprofloxacin are used to treat plague. Administration of anti-plague immunoglobulin, bacteriophage therapy.
Specific prophylaxis	Live-attenuated EV-type plague vaccine (Russia and Central Asian) or an inactivated whole-cell Y. pestis vaccine is recommended only for persons whose occupation regularly places them at high risk for exposure to Y. pestis or plague-infected rodents

ANTHRAX

Definition	Anthrax is an acute, deadly zoonotic disease caused by the bacterium Bacillus anthracis.
Etiology	Family – Bacillaceae Genus – Bacillus Species - B. anthracis
Morphology	Bacillus anthracis is a rod-shaped, Gram-positive, Capsulated, can form spore.
Epidemiology	**Source of infection**: infected animals (goats, cattle, sheep, horses…) or tissue from infected animals **Mode of transmission** (The mode of transmission depends on the type of anthrax): • direct or indirect contact • ingestion of raw or undercooked meat from an infected animal (marmots or prairie dogs, goat and camel) • airborne transmission • vector-borne transmission
Virulence factors	Exotoxin system (1) consisting of three separate proteins: protective antigen (PA'), edema factor (EF), and lethal factor (LF). Capsul.
Clinical forms	Cutaneous Inhalational Gastrointestinal
Methods of laboratory diagnostics	**Bacterioscopy** - direct Gram stained smear **Bacteriological method**: • isolation on culture media: MPA, MPB. • Identification: based on morphological, and biochemical properties; After cultivation in medium with penicillin, the pathogens acquired a spherical shape and resemble a pearl necklace. **Serological tests**: Termoprecipitation test (Ascoli test), ELISA, Immunofluorescence reaction **Allergological methods***:* anthraxin skin test **Molecular Genetic Methods:** PCR.
Treatment	Early antibiotic treatment of anthrax is essential—delay significantly lessens chances for survival. Treatment for anthrax infection includes large doses of intravenous and oral antibiotics, such as fluoroquinolones (like ciprofloxacin), doxycycline, erythromycin, vancomycin, or penicillin. In possible cases of inhalation anthrax, early antibiotic prophylaxis treatment is crucial to prevent possible death. Anthrax Immunoglobulin.
Specific prophylaxis	Live anthrax vaccine СТИ. Anthrax Vaccine Adsorbed (AVA, trade name BioThrax) is produced from culture filtrates of an avirulent, nonencapsulated strain known as V770-NP1-R.[3] No living organisms are present in the vaccine. Anthrax Immunoglobulin

TULAREMIA

Definition	Tularaemia is an acute, febrile, granulomatous, infectious zoonosis caused by the bacterium Francisella tularensis.
Etiology	Family – Enterobacteriaceae Genus – Francisella Species - F. tularensis
Morphology	F.tularensis is small, capsulated, non-motile and Gram-negative pleomorphic coccobacillus. Non spore forming.
Epidemiology	**Source of infection**: wild mammals (eg, rabbits, hares, prairie dogs, and muskrats, rats, voles, and other rodents); at least 9 species of domestic animals (eg, sheep, cattle, and cats); **Mode of transmission** Vector-borne transmission (Bites by infected arthropods) Direct contact with infected animals Handling of infectious animal tissues or fluids Ingestion of contaminated food, water, or soil Inhalation of infectious aerosols **Person-to-person transmission has not been documented.**
Virulence factors	The virulence determinants are not well known. Endotoxin. Fibrinolysin.
Clinical forms	Glandular (regional lymphadenopathy with no ulcer) Ulceroglandular (cutaneous ulcer with regional lymphadenopathy) Pneumonic (primary pleuropulmonary disease) Oculoglandular (conjunctivitis with preauricular lymphadenopathy) Oropharyngeal (stomatitis or pharyngitis or tonsillitis and cervical lymphadenopathy) Intestinal (intestinal pain, vomiting, and diarrhea) Typhoidal (febrile illness without early localizing signs and symptoms)
Methods of laboratory diagnostics	1. **Bacterioscopy** Direct stains for bacterial micromorphology (Tiny, faintly staining, pleomorphic gram-negative rods) 2. Serological methods: direct fluorescent antibody (DFA) stain; slide agglutination, antibody detection. 3. Biological (mouse inoculation) 4. Allergological methods: skin test with Tularin 5. PCR
Treatment	Streptomycin, Gentamicin, Ciprofloxacin, Doxycycline, Tetracycline and Chloramphenicol..
Specific prophylaxis	Live vaccine.

	BOTULISM	TETANUS
Definition	Botulism is a serious, but rare, paralytic illness caused by neurotoxins (botulinum toxin) produced by the common bacterium, Clostridium botulinum, which is found throughout the world in soil and ocean sediment.	Tetanus is a nervous system disorder characterized by muscle spasms that is caused by an exotoxin (tetanospasmin) produced by Clostridium tetani.
Etiology	Family – Bacillaceae Genus – Clostridium	
	Cl. botulinum	Cl. tetani
Morphology	Large anaerobic Gram-positive, motile, sporing rod bacteria that can only be cultured anaerobically. Non capsulated	
	Clostridium botulinum forms subterminal endospores	Clostridium tetani forms terminal endospores
Epidemiology	Botulism is not transmitted from person to person. ***Foodborne botulism*** *is caused by ingestion of food or drink containing botulinum toxin. Often this occurs when canned foods that are contaminated with C. botulinum are not adequately sterilized. The bacteria produce toxin while growing in the anaerobic interior of the can.*	Reservoir: Soil and intestine of animals and humans. Transmission: Contaminated wounds &tissue injury Temporal pattern: Peak in summer or wet season. Communicability: Not contagious
Virulence factors	The very strong botulinum neurotoxin is a heat-labile protein. Types A, B, and E cause poisoning in humans. The toxin is a metalloprotease that catalyzes the proteolysis of components of the neuroexocytosis apparatus in the motor end plates, resulting in flaccid paralysis of the musculature.	Clostridium tetani produces two exotoxins: a. Tetanolysin b.Tetanospasmin-a neurotoxin
CLINICAL FEATURES	Classic triad of botulism: • acute, symmetric, descending flaccid paralysis with prominent bulbar palsies • no fever • clear sensorium Features of bulbar palsy present in >90% patients: diplopia , dysarthria, dysphonia, dysphagia (difficulty with seeing, speaking and swallowing). Blurry vision with ptosis, diplopia, enlarged pupils with sluggish	Incubation period ranges from 3 to 21 days . Three clinical forms are recognized; Local Tetanus, in which there is contraction of muscles in the same anatomic area of injury; Cephalic tetanus occurring following the otitis media, head injuries. Generalised tetanus, which presents in a descending order, trismus, stiffness of neck, difficulty in swallowing, rigidity of abdominal muscles, temperature elevation, sweating.

	reactions. Death results from airway obstruction and respiratory muscle paralysis. Gastrointestinal symptoms present in foodborne botulism- abdominal cramps, nausea, vomiting, diarrhea.	
Methods of laboratory diagnostics	Based on toxin detection by means of the mouse neutralization test.	The preferred method is toxin detection in wound material in an animal test (mouse) based either on neutralization or detection of the toxin gene with PCR.
Treatment	Administration of botulinum antitoxin as soon as possible after diagnosis. The antitoxin is most effective if administered within 24 to 48 hours after the onset of symptoms	All wounds should be cleaned with removal of necrotic tissue &foreign material. Tetanus toxoid and human tetanus immune globulin should be given depending upon the vaccination status of the patient.
Specific prophylaxis		DT, or diphtheria-tetanus toxoids vaccine, protects against diphtheria and tetanus. DTP is a combined vaccine against diphtheria, tetanus, and pertussis.

TUBERCULOSIS

Definition	Tuberculosis is a common infectious disease caused by various strains of mycobacteria, usually Mycobacterium tuberculosis that most often affect the lungs.
Etiology	Family Mycobacteriaceae Genera Mycobacterium Species M. tuberculosis; M. bovis; M. avium.
Morphology	Mycobacterium tuberculosis is a slender, fairly, large, nonmotile, rod-shaped bacterium. Non-sporing. Staining: Ziehl-Neelsen stain.
Epidemiology	Source of infection: infected people, animals (sheep, cattle) **Mode of transmission** TB is primarily an **airborne** disease. The bacteria are spread from person to person in tiny microscopic droplets when a TB sufferer coughs, sneezes, speaks, sings, or laughs. Only people with active TB can spread the disease to others. Direct **contact. Ingestion** of contaminated food
Virulence factors	Chemical components of cell may play role in virulence. The cell wall is composed of mycolic acids, complex waxes, and unique glycolipids. Other important wall components are trehalose dimycolate (so-called cord factor, as it is thought to induce growth in serpentine cords on artificial medium) and mycobacterial sulfolipids. Another unique constituent is lipoarabinomannan (LAM). Hemolysins and lipases are produced by M. tuberculosis.
CLINICAL FEATURES	Patients usually present with pulmonary disease; prominent symptoms are chronic, productive cough, low-grade fever, night sweats, easy fatigability, and weight loss. Tuberculosis may present with or also exhibit extrapulmonary manifestations including lymphadenitis; kidney, bone, or joint involvement; meningitis; or disseminated (miliary) disease. Lymphadenitis and meningitis are more common among normal infants with tuberculosis.
Methods of laboratory diagnostics	**Bacterioscopic method:** Smears may be prepared from specimen like sputum, laryngeal swab, pleural fluid, peritoneal fluid, cerebrospinal fluid, pus, urine, gastric lavage, feces and other infected material. The smear is stained by Ziehl Neelsen technique. Acid fast bacilli are seen as pink brightened rods, while background is blue. **Bacteriological investigations**: isolation on culture media: Löwenstein–Jensen medium and Identification. **Allergological method** Skin Testing is performed as the tuberculin or Mantoux test test. PPD (purified protein derivative) is employed as the test antigen in the Mantoux test. The test is read within 48-72 hours. Different tests: PCR, Commercial chemiluminescent DNA probes, gas-liquid chromatography.
Treatment	First-line drugs: Isoniazid, Ftivazid, Rifampicin, Pyrazinamide, Ethambut Second-line drugs: Streptomycin, Ethionamide, Capreomycin, Cycloserine
Specific prophylaxis	Live, attenuated **vaccine BCG** (bacilli Calmette-Guerin)

DIPHTHERIA

Definition	Diphtheria is an acute respiratory tract infection caused by the bacteria Corynebacterium diphtheriae, a facultative anaerobic, Gram-positive bacterium. It is characterized by sore throat, fever, and an adherent membrane (a pseudomembrane) on the tonsils, pharynx, and/or nasal cavity. A milder form of diphtheria can be restricted to the skin. Less common consequences include myocarditis and peripheral neuropathy.
Etiology	Family Actinomycetacae Genera Corynebacterium Species C. diphtheria Corynebacterium diphtheriae is classified into biotypes (mitis, intermedius, and gravis) according to cultural properties, biochemical activity, antigenic structure etc.
Morphology	C. diphtheriae is nonmotile, noncapsulated pleomorphic Gram-positive rods. These divide by "snapping fission", so that adjacent cells lie at different angles to each other forming V-, L- and W-shapes. Staining: Some strains contain volutin granules. Methods of staining: Gram method, Neisser's method, Loeffler's methylene blue.
Epidemiology	Source of infection: sick people, carriers Transmission is droplet spread from the respiratory tract. More rarely transmission can occur from contact with articles soiled with discharges from infected lesions.
Virulence factors	Diphtheria exotoxin consists of hystotoxin, dermonecrotoxin, hemolisinum. The toxin acts locally on the mucous membranes of the respiratory tract to produce a grey, adherent pseudomembrane consisting of fibrin, bacteria, and epithelial and phagocytic cells. After absorption into the bloodstream, it acts systemically on the cell of the myocardium, the nervous system and the adrenal glands. The toxin can be rendered non-toxic but still antigenic by treatment with formaldehyde: the toxoid so formed is used in prophylactic immunization. Enzymes: Neiraminidase; Hyaluronidase; Factor of necrosis; Factors of diffusion.
CLINICAL FEATURES	The incubation period is a 2-5 day . There are two types of clinical diphtheria: nasopharyngeal and cutaneous. Early symptoms of pharyngeal diphtheria include a sore throat and fever, but after a few days a bluish-white adherent membrane, the pseudomembrane, forms over the back of the throat and tonsils.

Methods of laboratory diagnostics	**Specimens:** swabs from nose, throat, lessions **Bacterioscopic method**: Staining: Loeffler's methylene blue, Gram's, Albert's and Neisser's. Characteristic: dark purple volutin granules Arrangement: angled pairs, parallel rods (palisades), chinese letering **Bacteriological method** • For primary isolation, a variety of media may be used: Loeffler agar, Mueller-Miller tellurite agar, or Tinsdale tellurite agar. • Following initial isolation, C diphtheriae may be identified as mitis, intermedius, or gravis biotype on the basis of carbohydrate fermentation patterns and hemolysis on sheep blood agar plates. • The toxigenicity of C diphtheriae strains is determined by a variety of in vitro and in vivo tests. Some isolates of C.diphtheriae, especially mitis strains, are not toxigenic and are therefore non-virulent. The most common in vitro assay for toxigenicity is the immunodiffusion test. This test is based on the double diffusion of diphtheria toxin and antitoxin in an agar medium. **Biological method**: Guinea pig inoculation: inject subcutaneously a suspension of the isolated strain of C.diphtheriae into two guinea pigs, one protected with diphtheria antitoxin. If the strain is toxigenic, the upprotected animals dies in 2-3 days but the protected animal survives. **Serological methods**: ELISA; **PCR assays** (test presence of specific bacteriophate gene (tox) **Schick test:** a skin test formerly used to demonstrate immunity, i.e. circulating diphtheria antitoxin.
Treatment	Administration of diphtherial antitoxin serum (Ig) “Diapherm” Antibiotics (e.g., penicillin and erythromycin) are used as part of the treatment of patients who present with diphtheria.
Specific prophylaxis	In non-immune person (Schick positive) immunity can be produced by active or passive immunization. For active immunization: • DT, or diphtheria-tetanus toxoids vaccine, protects against diphtheria and tetanus. • DTP is a combined vaccine against diphtheria, tetanus, and pertussis. Passive immunity: antitoxin (anti diphtheria serum) is given subcutaneously. Being a horse serum one should take precaution against hypersensitivity.

PERTUSSIS

Definition	Pertussis, also known as whooping cough, is a highly contagious respiratory disease. It is caused by the bacterium Bordetella pertussis.
Etiology	Genera Bordetella Species B. pertussis
Morphology	Small, Gram-negative coccobacillus that appears singly or in pairs. It is non-motile, non-sporing, have a capsule..
Epidemiology	**Source of infection**: sick people, carriers **Mode of transmission: airborne** (spread by breathing in droplets expelled by an infected person when they talk, cough or sneeze).
Virulence factors	B. pertussis produces a variety of substances with toxic activity in the class of exotoxins and endotoxins (LPS). • invasive adenylate cyclase. This toxin acts locally to reduce phagocytic activity and probably helps the organism initiate infection; • lethal toxin (formerly called dermonecrotic toxin) which causes inflammation and local necrosis adjacent to sites where B. pertussis is located; • tracheal cytotoxin which is toxic for ciliated respiratory epithelium. The tracheal cytotoxin is a peptidoglycan fragment, which appears in the extracellular fluid where the bacteria are actively growing. The toxin kills ciliated cells and causes their extrusion from the mucosa. It also stimulates release of cytokine IL-1, and so causes fever; • pertussis toxin, PTx, a protein that mediates both the colonization and toxemic stages of the disease. Adherence mechanisms of B. pertussis involve a "filamentous hemagglutinin" (FHA), which is a fimbrial-like structure on the bacterial surface.
Clinical Features	Incubation period 7 – 10 days. Pertussis is divided into three stages. (1) The catarrhal stage, so named because of the mucous membrane inflammation, which is insidious and resembles the common cold. (2) Prolonged coughing sieges characterize the paroxysmal stage. During this stage the infected person tries to cough up the mucous secretions by making 5 to 15 rapidly consecutive coughs followed by the characteristic whoop—a hurried deep inspiration. The catarrhal and paroxysmal stages last about 6 weeks. (3) Final recovery may take several months (the convalescent stage).
Methods of laboratory diagnostics	Specimens: swabs from nose, throat **Bacteriological method** • For primary isolation may be used Bordet-Gengou medium • Identification: based on morphological, biochemical, cultural and antigenic properties; **Serological tests:** • fluorescent antibody staining of smears from nasopharyngeal swabs • agglutination reaction • complement fixation test

	• direct hemagglutination reaction).
Treatment	Treatment is with erythromycin, tetracycline, or chloramphenicol.
Specific prophylaxis	Prevention is with the DPT vaccine

SPIROCHETOSIS

	Syphilis	**Leptospirosis**
Definition	Syphilis is a sexually transmitted infection caused by the spirochete bacterium Treponema pallidum subspecies pallidum. The primary route of transmission is through sexual contact; it may also be transmitted from mother to fetus during pregnancy or at birth, resulting in congenital syphilis.	Leptospirosis is caused by infection with bacteria of the genus Leptospira and affects humans as well as other animals. Humans become infected through direct contact with the urine of infected animals or with a urine-contaminated environment. The bacteria enter the body through cuts or abrasions on the skin, or through the mucous membranes of the mouth, nose and eyes. Person-to-person transmission is rare.
Etiology	Family Spirochaetaceae	
	Genus – Treponema Species- T. pallidum	Genus –Leptospira Species L. interrogans
Morphology	Spiral-shaped, non-sporulating, motile, can't classify as gram (-) or (+) b/c don't stain well staining: biological stains (Borrelia), silver, Romanovsky-Giemsa stain, dark field microscopy, fluorescent microscopy	Spiral-shaped, non-sporulating, motile staining: stained with fluorescein-labelled antibodies Romanovsky-Giemsa stain dark field microscopy
Epidemiology	**Source of infection**: sick people **Mode of transmission**: (1) sexual contact (doesn't survive well outside host) (2) congenital (crosses placenta or contact while passing through birth canal, White Pneumonia) (3) blood transfusions (4) direct innoculation (contact w/secondary lesions in mouth by dentists)	**Source of infection:** infected animals (rodents, dogs, goats, pigs, cattle, sheep, …). **Mode of transmission:** • **Direct Contact** with the urine or tissues of infected animals. It can also be transmitted to humans who are in contact with water, soil or mud contaminated with infected urine, or who. The bacteria enter the body through cuts or abrasions on the skin, or through the mucous membranes of the mouth, nose and eyes). • **Ingestion** contaminated food or water • Human-to-human transmission occurs rarely (e.g., sexual transmission, transplacental transmission, transmission via breast milk).
Clinical Features	**Primary syphilis**. People with primary syphilis will develop one or more sores. The sores resemble large round bug bites and are often hard and painless. They occur on the genitals or in or around the mouth somewhere between 10-90 days (average three weeks) after exposure. Even without treatment they heal without a scar	Incubation period is 5 to 14 days (range 2 to 30 days). The usual clinical presentation is fever, chills, headache, severe myalgia, (particularly of the calves, thighs and lumbar region) and conjunctival suffusion. Severity varies with the infecting serovar.

	within six weeks. **Secondary stage** The secondary stage may last one to three months and begins within six weeks to six months after exposure. People with secondary syphilis experience a rosy "copper penny" rash typically on the palms of the hands and soles of the feet. However, rashes with a different appearance may occur on other parts of the body, sometimes resembling rashes caused by other diseases. They may also experience moist warts in the groin, white patches on the inside of the mouth, swollen lymph glands, fever, and weight loss. Like primary syphilis, secondary syphilis will resolve without treatment. **Latent syphilis**. This is where the infection lies dormant (inactive) without causing symptoms. **Tertiary syphilis**. If the infection isn't treated, it may then progress to a stage characterized by severe problems with the heart, brain, and nerves that can result in paralysis, blindness, dementia, deafness, impotence, and even death if it's not treated.	Weil's (syndrome jaundice, renal failure, haemorrhage and myocarditis) Meningitis and meningoencephalitis Pulmonary haemorrhage and ARDS.
Methods of laboratory diagnostics	**Specimens**: serous fluid from a chancre, blood, serum **Bacterioscopy**: dark ground microscopy of serous fluid from a chancre may be used to make an immediate diagnosis **Serological tests:** • direct fluorescent antibody testing • Complement fixation (CF) Wasserman test (the test determines the presence or absence of an AB that is produced in response to the presence of a constituent of the membrane of Treponema pallidum) • Treponema pallidum immobilization reaction (TPI) • The treponema pallidum particle agglutination assay (also called TPHA test)	**Specimens:** blood, serum, urine, liquor **Bacterioscopy**: dark ground microscopy, immunofluorescence or light microscopy ***Serological tests:*** • Radioimmunoassay (RIA) • Enzyme-linked immunosorbent assay (ELISA) • Microscopic agglutination test (MAT) • Complement fixation (CF) test **PCR assays** **Biological method** include the use of laboratory animals (Guinea pig, hamster).
Treatment	Penicillin G, Azithromycin, Cephalosporins, Doxycycline, Tetracycline	Penicillin G, Azithromycin, Cephalosporins, Doxycycline, Tetracycline
Specific prophylaxis		Human inactivated vaccines are available.

INFLUENZA

Definition	Influenza (also known as the flu) is an acute contagious respiratory infection caused by Orthomyxoviruses A, B, and C, occurs in local outbreaks, epidemics and pandemics.
Etiology	Influenza viruses belong to the Orthomyxoviridae family and are divided into types A, B and C. All influenza viruses are RNA viruses
Morphology	• The influenza viruses are spherical • The central core contains the viral RNA genome. • As in all viruses, the genome of an influenza virus particle is encased in a capsid that consists of protein. • Viral envelope covering protein capsid • The hemagglutinin (HA) and neuraminidase (NA) external glycoprotein antigens of epidemiologically important influenza viruses
Epidemiology	**Source of infection**: sick people Humans can become ill when infected with viruses from animal sources, such as avian influenza virus subtypes H5N1 and H9N2 and swine influenza virus subtypes H1N1 and H3N2. **Mode of transmission**: Droplet transmission (Influenza is spread via respiratory secretions (coughing and sneezing)) Airborne transmission
Clinical Features	**Incubation period** (the time from infection to illness) is about two days Fever, chills, cough, sore throat, runny or stuffy nose, muscle or body aches, headaches, fatigue.
Methods of laboratory diagnostics	**Virological method** • Isolation of influenza viruses on chicken embryos • Virus isolation in cell culture • Identification of influenza isolates by Hemagglutination inhibition test or Neutralisation Assay. **Serological tests:** • direct fluorescent antibody testing • ELISA assay **PCR assays**
Treatment	Antiviral drugs: Amantadine; Rimantadine; Zanamivir, Oseltamivir, Reaferon. Human immunoglobulin therapy
Specific prophylaxis	**Inactivated influenza vaccine** There are three types of inactivated vaccines, the ***whole virus vaccines***, ***split virus vaccines***, and ***subunit vaccines***. **Live, attenuated influenza vaccines**

MEASLES

Definition	Measles is a highly contagious viral respiratory disease, which affects mostly children and caused by a virus of the Paramyxovirus family. The disease is also called rubeola.
Etiology	Family – Paramixoviridae Genus Morbillivirus RNA viruse
Morphology	**RNA enveloped virus** • The measles virus particles are pleomorphic, spherical structures • The central core contains the viral RNA genome. • The genome of a measles virus particle is encased in a capsid that consists of protein. • Viral envelope covering protein capsid
Epidemiology	**Source of infection**: sick people **Mode of transmission**: Droplet transmission (Measles is spread via respiratory secretions (coughing and sneezing)) Airborne transmission
Clinical Features	The incubation period from exposure to onset of measles symptoms ranges from 7 to 14 days (average, 10-12 days). There are two stages in measles. 1. Prodromal stage is marked by malaise, fever, redness of eyes, and the classic triad of conjunctivitis, cough, and coryza (with sneezing and nasal discharge). Koplik's spots appear on the buccal mucosa opposite the first and second upper molars. They are small, bluish white spots on a red base, smaller than the head of a pin. 2. Eruptive stage or rash stage Typical dusky red rash which begins behind the ears, and within a few hours spreads over the face and neck, and slowly extends down the body
Methods of laboratory diagnostics	**Virological method** • Virus isolation in cell culture • Identification of measles isolate by Neutralisation Assay. **Serological tests**: • direct fluorescent antibody testing • ELISA assay **PCR assays**
Treatment	There is no treatment that can kill the measles virus, so treatment focuses on supportive care, or the relief of symptoms.
Specific	**Live, attenuated measles vaccines.** The MMR vaccine is an immunization shot against **measles**, mumps, and rubella. The vaccine is a mixture of three live attenuated

prophylaxis	viruses, administered via injection. **Inactivated measles vaccine** **Human Normal Immunoglobulin**

RUBELLA

Definition	Rubella is a highly contagious viral disease characterized by slight fever, mild rash and swollen glands. Although rubella causes only mild symptoms of low fever, swollen glands, joint pain, and a fine red rash in most children and adults, it can have severe complications for women in their first trimester of pregnancy. These complications include severe birth defects or death of the fetus.
Etiology	Family – Togaviridae RNA viruse
Morphology	**RNA enveloped virus** The measles virus particles are pleomorphic, spherical structures • The central core contains the viral RNA genome. • The genome of a rubella virus particle is encased in a capsid that consists of protein. • Viral envelope covering protein capsid Rubella virus contains one capsid protein and three envelope glycoproteins
Epidemiology	**Source of infection**: sick people **Mode of transmission**: • Droplet transmission • Vertical transmission from mother to child. Rubella virus can cross the placenta and establish infection in the developing fetus, causing congenital Rubella syndrome (CRS).
Clinical Features	The average incubation period is 14 days, with a range of 12–23 days. Rubella usually presents as a nonspecific, maculopapular, generalized rash with generalized lymphadenopathy. When rubella virus infection occurs during early pregnancy, consequences may include miscarriage, fetal death, or an infant born with the constellation of severe birth defects known as congenital rubella syndrome. The most common congenital defects are cataracts, heart defects, and hearing impairment.
Methods of laboratory diagnostics	**Virological method** • Virus isolation in cell culture • Identification of measles isolate by Neutralisation Assay. **Serological tests:** • direct fluorescent antibody testing • ELISA assay **PCR assays**
Treatment	There is no specific antiviral therapy for rubella; basic treatment consists of supportive care.

Specific prophylaxis	Inactivated rubella vaccine Live, attenuated rubella vaccine

Hepatitis A, B

	Hepatitis A	Hepatitis B
Definition	Hepatitis A is an acute infectious disease of the liver caused by the hepatitis A virus, an RNA virus, usually spread by the fecal-oral route; transmitted person-to-person by ingestion of contaminated food or water or through direct contact with an infectious person.	Hepatitis B is an acute or chronic viral infection that attacks the liver, caused by the hepatitis B virus. It can cause chronic liver disease and puts people at high risk of death from cirrhosis of the liver and liver cancer.
Etiology	Family - Picornaviridae	Family - Hepadnaviridae
Morphology	RNA non enveloped viruses • The central core contains the viral RNA genome. • The genome of a hepatitis A virus particle is encased in a capsid	DNA enveloped virus • The central core contains the viral DNA genome. • The genome of a Hepatitis B virus particle is encased in a capsid that consists of protein. • Viral envelope covering protein capsid Antigens: HBs-Ag is the surface antigen of the hepatitis B virus. HBc-Ag (core antigen) is a hepatitis B viral protein. It is an indicator of active viral replication; HBe-Ag is the extracellular form of HBcAg
Epidemiology	**Source of infection**: Infected individuals **Mode of transmission:** • fecal-oral (contaminated food or water)	**Source of infection**: Infected individuals, carriers of hepatitis B virus **Mode of transmission**: • blood transfusions and transfusion with other human blood products • re-use of contaminated needles and syringes • sexual contact • vertical transmission from mother to child during childbirth. s
Clinical Features	Asymptomatic Symptomatic without jaundice Symptomatic with jaundice	Most people do not experience any symptoms during the acute infection phase. However, some people have acute illness with symptoms that last several weeks, including yellowing of the skin and eyes (jaundice), dark urine, extreme fatigue, nausea, vomiting and abdominal pain. In some people, the hepatitis B virus can also cause a chronic liver infection that can later develop into cirrhosis of the liver or liver cancer

Methods of laboratory diagnostics	**Virological method** • Virus isolation in cell culture • Identification of measles isolate by Neutralisation Assay or by direct immunofluorescence method **Serological tests**: • direct immunofluorescence method • ELISA assay **PCR assays**	**Serological tests**: • ELISA assay (the detection of the hepatitis B surface antigen HBsAg) A positive test for the HBsAg indicates that the person has an active infection (either acute or chronic) • Direct fluorescent antibody testing **PCR assays**
Treatment	There is no specific treatment for hepatitis A. Sufferers are advised to rest, avoid fatty foods and alcohol, eat a well-balanced diet, and stay hydrated.	There is no specific treatment for acute hepatitis B. Care is aimed at maintaining comfort and adequate nutritional balance, including replacement of fluids that are lost from vomiting and diarrhoea. Some people with chronic hepatitis B can be treated with drugs, including interferon and antiviral agents.
Specific prophylaxis	**Passive** immunization with serum immunoglobulin **Active**– Inactivated and live attenuated vaccines against hepatitis A	Inactivated Hepatitis B Vaccine (**Recombinant**)

POLIOMYELITIS

Definition	Poliomyelitis is an enteroviral infection that can manifest in 4 different forms: inapparent infection, abortive disease, nonparalytic poliomyelitis, and paralytic disease. Poliomyelitis is caused by a picomavirus (very small RNA virus).
Etiology	Family Picomaviridae Genus Enterovirus
Morphology	RNA non enveloped viruses The central core contains the viral RNA genome. • The genome of a poliovirus particle is encased in a capsid
Антигенная структура	Три антигенах типа: 1, 11 и 111. В период эпидемических вспышек доминирует 1 тип, вызывающий паралитическую форму заболевания
Source of infection	Infected individuals
Mode of transmission	• the major route of transmission is fecal-oral (contaminated food or water) also can be transmitted through droplet spread of respiratory secretions
Clinical Features	There are 4 different clinical forms of Poliomyelitis: inapparent infection, abortive disease, nonparalytic poliomyelitis, and paralytic disease.
Clinical Features	There are 4 different clinical forms of Poliomyelitis: inapparent infection, abortive disease, nonparalytic poliomyelitis, and paralytic disease
Methods of laboratory diagnostics	Virological method • Virus isolation in cell culture • Identification of poliovirus isolate by Neutralisation Assay or by direct immunofluorescence method Serological tests: • direct immunofluorescence method ELISA assay PCR assays
Treatment	There is no specific treatment for polio.
Specific prophylaxis	Live attenuated oral polio vaccine (OPV) developed by Dr. Albert Sabin in 1961. Inactivated (killed) polio vaccine (IPV), developed in 1955 by Dr Jonas Salk.

RABIES

Definition	Rabies is a zoonotic viral disease affecting the nervous system. The disease infects domestic and wild animals, and is spread to people through close contact with infected saliva via bites or scratches.
Etiology	Family Rhabdoviridae Genus Lissavirus
Morphology	RNA enveloped virus; bullet shape; genome of a virus particle is encased in a capsid that consists of protein. Viral envelope covering protein capsid.
Антигенная структура	Два антигена: s -антиген внутренний (нуклеопротеидный) v-антиген в составе суперкапсида (гликопротеидный)
Source of infection	all warm-blooded animals can be infected. The disease is found most commonly in dogs, foxes, cats, raccoons.
Mode of transmission	Rabies is spread by exposure to the saliva of a rabid animal through a bite. On rare occasions, it can also be transmitted by a scratch from an infected animal or by virus-laden saliva coming into contact with a fresh break in the skin or intact mucus membranes (eyes, nose, mouth).
Clinical features	The average duration of incubation is 20-90 days. Rarely, incubation has been reported up to 7-19 years. *Prodromal period* The virus enters the CNS. Nonspecific symptoms and signs develop. Paresthesia, pain, or intense itching at the inoculation site is pathognomonic for rabies Symptoms may include the following: Malaise, Anorexia, Headaches, Fever, Chills, Insomnia, Depression. *Acute neurologic period* This period is associated with objective signs of developing CNS disease. Symptoms include muscle fasciculations, priapism, and focal or generalized convulsions. Patients may die immediately or may progress to paralysis, which may be present only in the bitten limb at first but usually becomes diffuse. Two clinical forms of rabies are recognize: 1) *a. furious form* associated with classical signs of excitation or phobic symptoms, 2) *Dumb rabies (paralytic rabies)* characterized by progressive paralysis without an initial furious phase.

Methods of laboratory diagnostics	*Diagnosis in humans* Several tests are necessary to diagnose rabies ante-mortem (before death) in humans. Samples of saliva, serum, spinal fluid, and skin biopsies of hair follicles at the nape of the neck. Tests: **polymerase chain reaction** (RT-PCR), **ELISA** Post mortem, the standard diagnostic technique is to detect rabies virus antigen in brain tissue by **fluorescent antibody test.**
Treatment	There is no treatment for rabies that can cure the disease once symptoms appear. Post-exposure treatment for rabies: • local treatment of the wound • rabies immune globulin the rabies vaccine
Specific prophylaxis	Rabies is a vaccine-preventable disease. The most effective strategy for preventing rabies in people is by eliminating rabies in animals (dogs, cats) through vaccination. Preventive immunization in people. Pre-exposure immunization in people is recommended for travellers to high-risk areas in rabies-affected countries, and for people in certain high-risk occupations such as laboratory workers dealing with live rabies virus and other lyssaviruses, and veterinarians and animal handlers in rabies-affected areas.

HIV

Definition	The infection is caused by the human immunodeficiency virus (HIV).
Etiology	Family Retro viridae Genus Lentivirinae
Morphology	Enveloped RNA virus spherical in shape Genome of a HIV particle is encased in a capsid that consists of protein. Viral envelope covering protein capsid. HIV envelope proteins include gp 41, gp 120, and gp 160. Reverse transcriptase (RT), protease (PR) and integrase (IN) are three key enzymes of HIV. There are two types of HIV: HIV-1 & HIV-2 HIV affects the immune system. HIV infects cells that have the CD4 receptor on their cell membrane (T helper).
Антигенная структура	Представлена белками и гликопротеидами. Отличается очень высокой изменчивостью.
Source of infection	Infected individuals, carriers of human immunodeficiency virus
Mode of transmission	Mode of transmission: • blood transfusions and transfusion with other human blood products • re-use of contaminated needles and syringes • sexual contact vertical transmission from mother to child during childbirth.
Clinical features	There are four stages of HIV infection: primary, asymptomatic, symptomatic, and progression from HIV to AIDS (acquired immune deficiency syndrome).

Methods of laboratory diagnostics	**Serological Methods:** Detection of HIV antibody * Screening tests: **ELISA** * Supplemental tests: **Western Blot (immunoblot)** Indirect Immunofluorescence Radio Immunoprécipitation Assay **Detection of viral nucleic acid:** PCR, RT-PCR
Treatment	**Antiretroviral therapy (ART)** consists of the combination of at least three antiretroviral **(ARV)** drugs to maximally suppress the **HIV** virus and stop the progression of **HIV** disease. *Nucleoside/Nucleotide Reverse Transcriptase Inhibitors (NRTIsj* lamivudine and zidovudine *Nonnucleoside Reverse Transcriptase Inhibitors (NNRTIs):* rilpivirine, delavirdine *Protease Inhibitors (Pis):* indinavir, ritonavir *Fusion Inhibitors:* enfuvirtide **Immunomodulating therapy Symptomatic therapy**
Specific prophylaxis	There is no effective vaccine

CANDIDIASIS

Definition	Fungal infection, manifested by superficial and profound lesions of mucous membranes, skin and internal organs.
Etiology	Imperfect fungi are deuteromycetes. Yeast-like fungi of the genus Candida species: C. albicans, C. tropicalis, C. pseudotropicalis, C. krusei
Morphology	Unicellular microorganisms of oval form, form pseudomycelia, blastospores. Stained with methylene blue, according to Gram, Romanovsky-Giemsa.
Factors of virulence	Adhesiveness, secretion of proteolytic enzymes, hemolysins. Endotoxins.
Source of infection	Most often, candidiasis occurs as an endogenous infection.
Methods of laboratory diagnostics	1. Microscopic examination of smears stained by Gram or methylene blue. 2. Mycological studies - isolation of pure fungal culture on Saburo agar, wort agar or candida agar, followed by identification by morphological, cultural, biochemical properties. 3. Serological studies - determination of the titer of AT in the blood serum: RA, RNGA, RSK, RP. 4. Allergodiagnostics - intradermal test with candidal allergens. 5. Biological method - determination of pathogenicity of Candida on white mice.
Treatment	Antifungal antibiotics: nystatin, levorin, miconazole, griseofulvin, amphotericin B.
Specific prophylaxis	Not developed.

	Trichophytia	**Microsporia**
Definition	Trichophytosis (ringworm) is a disease of the skin and its appendages (hair and nails) caused by various species of fungi of the genus Trichophyton.	Microsporia (synonym: microporos, ringworm) - contagious skin disease and hair caused by pathogenic fungi Microsporum. Infection occurs mainly from patients microsporia cats rarely dogs, in 20% of cases of sick people.
Etiology	Genus: - Trichophyton species *Trichophyton violaceum*, Tr. Gypseum	Genus: - Microsporum species - Microsporum canis
Morphology	characterized by the presence of thin, short, branching, segmented filaments of mycelium, contain chlamydospores. Branching at right angles is characteristic	Septic mycelium
Source of infection	Sick people and animals	
Mode of transmission	contact	
Methods of laboratory diagnostics	1. Microscopic method - the study of the morphology of the fungus under microscopic examination in 20-30% alkali. 2. Mycological method - isolation of the culture of the fungus on the Saburo medium with subsequent identification. 3. Skin-allergic tests with allergens from fungi.	
Treatment	Griseofulvin, clotrimazole, amphotericin B	
Specific prophylaxis	Absent	

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