STD’s and Animal Diseases

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Viral STD’s
HIV

Retroviral RNA
Attacks T cells
Envelop, capsid, DNA
Sex, blood, birth
HTLV-1 - first discovered retrovirus 1977
HIV Pathogenesis

GP160 attaches to CD4 receptor
  • T HELPER, monocytes, macrophages

Replication, processing, budding
  • Reverse transcriptase, Integrase, Protease

Gradual destruction of T-cells

AIDS diagnosed when T-cell count<200

Acute viral illness -> latency -> opportunistic infection
Associated with: Candida albicans, PCP, Kaposi’s Sarcoma, TB, EBV, CMV, Fungi

Diagnosis- ELISA, PCR, Western Blot

No Cure, or Vaccine- (rapid mutation)

• Antiretroviral drugs
Herpes Viruses

Lots of commonly seen diseases
dsRNA, enveloped
Latent State- reside in the sensory ganglia
Cell mediated immune response
Simplexvirus- direct contact of mucous membranes, birth
- HSV-1- cold sores, HSV-2- genital herpes
- Gingivostomatitis, genital herpes
Mononucleosis

EBV - Epstein-Barr Virus

Affects lymphocytes - fever, chills, sweats, painful pharyngitis, malaise.

• Invades B cells - inducing abnormal growth (cancer)
  • Associated with Burkitt’s lymphoma
• Monospot test for diagnosis
CMV- cytomegalovirus- named after swollen cells

- Congenital, milk, saliva, urine, tears, sex
- Diagnosis- Buffy Coat, PCR
Human Papilloma Virus

Circular dsDNA

Over 50 strains

Warts - skin, larynx, cervix, mucosa
  • Cannot bind live tissue - keratinocytes/epithelium

Cervical Cancer
  • Type 16, 18, 31, 33
  • CIN(dyslasia) I -> II -> III -> Carcinoma in Situ

Pap Smear

Vaccine - against 16, 18
Bacterial STDs

SHE MAY LOOK CLEAN—BUT

PICK-UPS
"GOOD TIME" GIRLS
PROSTITUTES

SPREAD SYphilis AND GONORRHEA

You can't beat the Axis if you get VD
Neisseria gonorrhoea

Gram negative, coffee bean shaped

Motile, grappling hook pilli

Same symptoms as Chlamydia: purulent discharge (dissemination to conjunctivitis, pharyngitis, endocarditis, asymptomatic, etc.)

Resistant to penicillin, use cephalosporin

Chocolate agar with CO₂, oxidase/catalase colistin positive → serological test
Chlamydia trachomatis

Obligate intracellular bacteria

Virus like life cycle: reticulate bodies/elementary bodies

Purulent genital discharge, cervicitis, urethritis, conjunctivitis, reactive arthritis, etc

PCR, ELISA diagnosis

Macrolides, tetracyclines
Treponema pallidum

Spirochete bacterium

Single lesions → multiple lesions → gummatous/neuro/cardiovascular syphilis

Primary, secondary, latent, tertiary stages (also congenital)

Identified by serology or special stains (Dieterle stain)

Penicillin G
Arboviruses
Arboviruses

Arthropod-borne viruses
RNA viruses
Transmitted by bloodsucking arthropods
• Ticks, fleas, mosquitoes

Arenaviridae
Bunyaviridae
Filoviridae
Reoviridae

• Flaviviridae
  ➢ Flavivirus
• Togaviridae
  ➢ Alphavirus
Disease

Alphavirus

- Eastern, Western, Venezuelan equine encephalitis

Flavivirus

- St. Louis Encephalitis
- West Nile Fever
- Japanese B Encephalitis
- Dengue
- Yellow Fever
Pathogenesis
(\textit{Flavivirus & Alphavirus Encephalitis})

Multiplication in myeloid, lymphoid, or vascular endothelium cells

Viremia for several days after infection

Vectors acquire virus by sucking blood

May or may not cross blood-brain barrier

Majority of infection is controlled before neuroinvasion occur

Susceptibility to CNS infection is age dependent in Human Equine encephalitides is diphasic

• Minor illness
• Major illness
Symptoms, Lab and Clinical Finding

Incubation period 4-21 days
Inapparent infections common
Flu like illness or encephalitis
Sudden onset of severe headache, chills, fever, nausea, vomiting, generalized pains
Within 1-2 days, marked drowsiness and stupor develops
Mental confusion, convolution, coma in severe cases
Mortality varies
Neutralizing and Hemagglutination inhibiting antibodies detected within a few days after illness
Pathogenesis

Yellow Fever

Introduced by mosquito through skin & multi.

Spread to lymph nodes, liver, kidney, bone marrow, myocardium

Viremia during early infection

Lesions are due to localization of virus in a particular organ

Can have hemorrhage and circulatory collapse
Clinical, Lab

Incubation 3-6 days

At onset, fever, chills, headache, dizziness, myalgia, backache – followed by nausea, vomiting, and bradycardia

After initial phase, 15% go on to severe phase

Fever, jaundice, renal failure, hemorrhage

Mortality 20% or more (esp. children, elderly)

Death occurs on day 7-10

No sequelae

IgM antibodies during first week of illness
**DENGUE**

- Incubation 4-7 days
- Onset of fever, chills, headache
- Pain in back, joints, muscle, eyeballs
- Fever last 2 to 7 days.
- Rash, Enlarged lymph node
- Death is rare

**Dengue shock syndrome/dengue hemorrhagic fever**

- Increased vascular permeability
- Plasma leakage into interstitial spaces
- Lead to shock
- Caused by preexisting dengue antibody from previous infection
IgM antibodies develop within few days

Neutralizing and hemagglutination-inhibiting antibodies appear within a week

Significant rise in antibody titer is sure sign of active dengue infection
Bacteria Related to Animals
Yersinia Pestis
(Bubonic Plague)
Y. pestis: Microbiological Characteristics

- Gram-negative
- Nonmotile
- Exhibits bipolar stains
- Anerobic: prefers blood or tissue fluids around 30°C
Y. pestis: Transmission & Pathogenesis

• Multiply in macrophages
• Produce antiphagocytic protein
  – resist phagocytosis
• Pathogens reach lymphatics
  – hemorrhagic inflammation develops in lymph nodes
  – Undergoes necrosis, becomes fluctuant
• Organism reach bloodstream
  – hemorrhagic and necrotic lesions develop in all organs
Y. pestis: Virulence Factors

Encodes two antigenic molecules: Fraction 1 (F1) capsular antigen, and VW antigen.

Both molecules needed for pathogenicity, not expressed lower than 37°C. Therefore, is not virulent in fleas, since their body temperature is around 25°C.

Is a model for studying Type III Secretion Systems (TTSS) that inject bacterial proteins into a host cell.

The translocation of Yersinia outer proteins (Yop’s) block the host cell’s ability to communicate with immune system cells and down-regulates the response of phagocytic host cells to infection.

The TTSS, YopH and Yersinia protein kinase A (YpkA) are delivered by YopB and YopD into the host cell, where they subvert signal transduction and inhibit oxidative bursts.

The rough/short lipopolysaccharide (LPS) chains on the outer membrane of Yersinia mediate antibody resistance by causing abnormal attachment of membrane attack complexes (MACs).
**Y. pestis: Clinical Aspects**

**Symptoms**
- Incubation period of 2–6 days
- Lack of energy
- Fever
- Headaches and chills
- Swelling of lymph nodes: BUBOES

**Treatments**
- Antibiotics
  - Streptomycin
  - Chloramphenicol
  - Gentamicin

**Diagnostic Tests**
- Specimen
- Smear
- Culture
- Serology
Francisella Tularensis
(Tularemia: Rabbit Fever)
F. tularensis: Microbio. Characteristics

Gram-negative bacteria
Coccobacillary shaped
Non-motile
Strict aerobes
Bacteria enters through skin abrasions
2-6 days, inflammatory ulcerating papule develops
Lymph nodes enlarge and become necrotic
Strains produce different hemolytic agents, which may facilitate degradation of the phagosome.

Acid phosphatase AcpA has been found in other bacteria to act as a hemolysin, whereas in Francisella its role as a virulence factor is under vigorous debate.

does not contain typical virulence secretion systems, but does contain a number of ATP binding cassette (ABC) proteins that may be linked to the secretion of virulence factors.

Uses type IV pili to bind to the exterior of a host cell and thus become phagocytosed.

The expression of IglC is required for phagosomal breakout and intracellular replication.

In vitro, it down-regulates the immune response of infected cells to ensure that replication is unhindered by the host immune system by blocking the warning signals from the infected cells.
F. tularensis: Clinical Aspects of Tularemia

Symptoms
- Ulceroglandular
- High fever
- Organ failure
- Malaise
- Headache
- Death (1%)

Treatments
- Streptomycin
- Gentamicin (10 days)
- Doxycycline (2-3 weeks)

Diagnostic Tests
- Serological tests
  - titer of 1:160
Rickettsia

Thyphus
Ricketsialpox
Boutonneuse fever
African tick bite fever
Rockey Mountain spotted fever
Flinders Island spotted fever
Queensland tick typhus
Pathogenesis

Rickettsiae are transmitted when a human is bit by an infected host, such as a tick or flea. If a mammal, such as a dog or cat, is bitten by an infected host, it can become a host as well and transmit to other mammals or people.
Symptoms

Skin lesions
Papulovesicular rash
Headache
Backache
Diagnosis

If clinical symptoms and the epidemiologic history are compatible with rickettsial infections, the following diagnostic tests should be used during the acute stage of illness and at the time antibiotic treatment is initiated:

- PCR test on skin biopsy of rash or eschar or EDTA whole blood
- Specific immunohistologic detection of rickettsiae in skin biopsy of rash or eschar
Leptospira

It causes the disease Leptospirosis.

In humans, Leptospirosis can cause a wide range of symptoms, including:

- High fever
- Headache
- Chills
- Muscle aches
- Vomiting
- Jaundice (yellow skin and eyes)
- Red eyes
- Abdominal Pain
- Diarrhea
- Rash
The bacteria that cause leptospirosis are spread through the urine of infected animals, which can get into water or soil and can survive there for weeks to months. Many different kinds of wild and domestic animals carry the bacterium.

Humans can become infected through:

- contact with urine (or other body fluids, except saliva) from infected animals
- contact with water, soil, or food contaminated with the urine of infected animals.

The bacteria can enter the body through skin or mucous membranes (eyes, nose, or mouth), especially if the skin is broken from a cut or scratch. Drinking contaminated water can also cause infection. Outbreaks of leptospirosis are usually caused by exposure to contaminated water, such as floodwaters. Person to person transmission is rare.
The diagnosis of leptospirosis is made by culture of the bacterial organism *Leptospira* from infected blood, spinal fluid, or urine. However, many doctors must rely upon rising *Leptospira* antibody levels in the blood in order to make the diagnosis, as the technique required to perform the culturing is delicate and difficult.
Prions

Proteinaceous Infectious Particles

CJD, kuru
  • Mad Cow Disease

Due to mutate PrP (Prion Protein)
  • PrPc (cellular) is normal
  • PrPsc (scrapie) is mutated

Long incubation time

Non-inflammatory
Pathogenesis

Can be inherited or acquired

Acquired
  • Exposure to PrPsc via beef consumption or contamination
  • PrPsc induces change in regular PrPc

Inherited
  • Chromosome 20
  • Post-translational error
Neurologic Symptoms

• Dementia
• Ataxia
• Psychiatric symptoms
• Death

There is no cure
<table>
<thead>
<tr>
<th>Microorganism</th>
<th>Disease</th>
<th>Signs/Symptoms</th>
<th>Virulence Factors/Pathogenesis</th>
<th>Clinical Features</th>
<th>Special Lab Diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>HIV</td>
<td>AIDS</td>
<td>Increased susceptibility to MANY diseases</td>
<td>Protease, Integrase, Reverse Transcriptase</td>
<td>Tcell count &lt; 200</td>
<td>Western Blot, ELISA, PCR</td>
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<tr>
<td>Herpesvirus</td>
<td>Herpes, Chicken Pox, Mononucleosis</td>
<td>HSV- cold sores, Gingivostomatitis EBV, CMV-pharyngitis, fever, malaise,</td>
<td>Immune Invasion MHCII downregulation</td>
<td></td>
<td>Tzanck Prep, PCR, viral culture, Direct fluorescent antibodies</td>
</tr>
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<td>HPV</td>
<td>Warts Cervical Carcinoma</td>
<td>Warty neoplasms on face, genitals, mucosal surfaces</td>
<td>E6- inactivates p53 E7-RB</td>
<td>Increase risk in smokers and immunodeficient</td>
<td>PAP smear to detect dysplasia</td>
</tr>
<tr>
<td>Nesseria Gonorrhea</td>
<td>Gonorrhea</td>
<td>Urethritis, PID, ectopic pregnancy, peritonitis</td>
<td>Pili for adherence, IgA protease</td>
<td>Can cause sterility</td>
<td>Gram stain, chocolate agar culture, PCR</td>
</tr>
<tr>
<td>Chlamydia Trachomatis</td>
<td>Chlamydia</td>
<td>Trachoma, conjunctivitis, pneumonia, PID</td>
<td>Lysozyme resistant, prevent phagolysosome formation</td>
<td>Cannot be grown in media</td>
<td>Immunofluorescence tests, serologic tests</td>
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<td>Treponema Pallidum</td>
<td>Syphilis</td>
<td>Chancre, rash, condyloma latum Progresses to severe CNS involvement</td>
<td>Very motlie</td>
<td>Can be contracted in utero Only 25% of latent relapse</td>
<td>ELISA, FTA-ABS, MHA-TP,</td>
</tr>
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<td>Alphavirus</td>
<td>Equine Encephalitis, Rubivirus</td>
<td>Headache, fever, arthritis</td>
<td>Mosquito vetor</td>
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<td>Detection of Antibodies in serum</td>
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<td>Flavivirus</td>
<td>Dengue fever, Yellow fever, West Nile</td>
<td>Hepatitis, Fever Dengue- can be hemorrhagic</td>
<td>Aedes, Culex</td>
<td>Travelers need vaccination</td>
<td>Viral culture, Serology</td>
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<td>Yersinia Pestis</td>
<td>Bubonic Plague (rodent reservoirs)</td>
<td>Lymph node swelling, fever, pneumonic, “black death”</td>
<td>F1, V and W proteins</td>
<td>Extremely high death rate</td>
<td>Gram Stain, blood culture, serology</td>
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<tr>
<td>Francisella Tularensis</td>
<td>Tularemia</td>
<td>Ulcerous gland at sight of contact, pneumonia, typhoidal</td>
<td>Antiphagocytotic Reservoirs in rabbits, ticks, deer</td>
<td>Highly infective (skin contact)</td>
<td>Skin test, IgG antibody titer</td>
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<tr>
<td>Rickettsiae</td>
<td>Rocky mountain spotted fever, epidemic typhus, pox</td>
<td>Rash, fever, headache, redness</td>
<td>Damages endothelial cells, reservoirs: rodents, dogs, birds</td>
<td></td>
<td>Weil-Felix reaction, OX19, OX2, OX-k reactions</td>
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<td>Leptospira</td>
<td>Weil’s Disease</td>
<td>Fever, muscle aches, meningismus, renal failure, hep</td>
<td>Zoonotic in dogs, cats, livestock. Contact with urine or animal tissue</td>
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<td>Culture CSF or blood, ELISA, PCR</td>
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<td>Prions</td>
<td>CJD, GSS, kuru</td>
<td>Dementia, ataxia, neuro symptoms</td>
<td>Inherited or from contact with cow PrPsc.</td>
<td>Long incubation time</td>
<td>Immunostaining for PrPsc of brain</td>
</tr>
</tbody>
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