SEXUALLY TRANSMITTED DISEASES

Syphilis

A systemic venereal disease caused by the spirochete *Treponema pallidum.* The organisms cannot be cultured but are detected by

1. Silver stains
2. Dark-field examination
3. Immunofluorescence.

Clinical features and pathogenesis

The primary stage

This occurs about 3 weeks post-infection, as a single firm non-tender red raised lesion (*chancre*) located on the external genitalia. It heals in few weeks without therapy. Microscopical examination shows intense plasma cell & macrophage infiltrate with an obliterative endarteritis. These form the hallmarks of histologic changes in all stages of syphilis

Secondary stage

This occurs 2-10 weeks after primary chancre, as a diffuse rash involving palms & soles mainly, with white oral lesions. There are in addition, fever, lymphadenopathy, headache & arthritis.

Tertiary stage

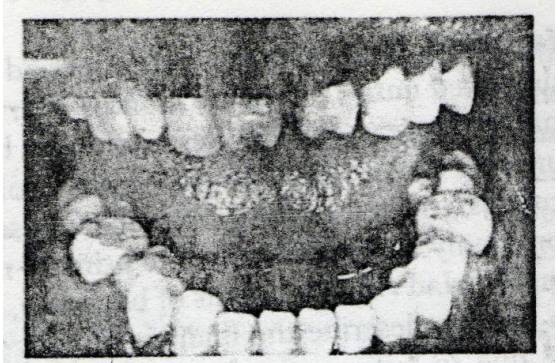
This occurs years later as active inflammatory lesions of aorta, heart & CNS or quiescent lesions in the liver, bones & skin called **gummas.**

On histologic examination, gummas contain a center of coagulated necrotic material surrounded by granulomas made of palisaded macrophages & fibroblasts & an outer layer of plasma cells.

Congenital syphilis

Treponemas from an infected mother invade the placental tissue of the fetus at about the 5^{th} month of gestation causing one of the following:

1- Late abortion,
2- Still birth,
3- Death after delivery
4- It may persist in a latent form to appear in childhood or adult life manifestations include Hutchinson teeth with peg like incisors, CNS manifestations, interstitial keratitis, bone deformities,



Congenital syphilis. The patient exhibits anomalously shaped teeth consisting of incisors with screwdriver-shaped crowns and notched incisal edges and molars with constricted crowns and a lack of cuspal development (mulberry molars).

<u>Herpes virus infections</u>

Herpes viruses are large encapsulated DNA viruses. **HSV-1 & HSV-2** are both neurotropic, replicate in skin & mucus membranes at the site of entrances of the virus (oropharynx or genitalia) causing vesicular lesions & infect neurons of the location.

In immune competent host, the primary infection resolves in few weeks & the virus remains latent in nerve cells. Reactivation leads to its spread again to skin & mucous membranes.

Pathological features

There is formation of large pink to purple intranuclear inclusions that contain intact & disrupted virions pushing cellular chromatin to the end of the nucleus. Cells form multinucleated syncytia containing inclusions are diagnostic in smears of blister fluid.

Clinical diseases

Fever blisters (cold sores)

Intraepithelial vesicles are formed by intracellular edema & ballooning degeneration of epithelial cells of skin around mucosal orifices (lips & nose). They may burst & crust or show superficial ulcerations. They are caused by HSV-1

Gingivostomatitis

This occurs in children & is caused by HSV-1. It is a vesicular eruption, which extends from the tongue to retropharynx with an associated cervical lymphadenopathy.

Genital herpes

Vesicles are seen on the genital mucous membranes & external genitalia leading to ulceration & rimmed by inflammation. It is caused by HSV-2 or 1.

Transmission to neonates occurs during delivery causing mild to fulminating infection with lymphadenopathy, splenomegaly & areas of necrosis in the liver, lungs, adrenal & CNS.

Ebola virus

Ebola virus is an RNA virus belonging to filovirus, it cause a hemorrhagic disease with high mortality rate in human in several regions of Africa.Infections with Ebola virus either (Zaire strain) and (Sudan strain) have cause an Ebola Hemorrhagic Fever which is a fetal African Disease. Recent field evidence has implicated several species of fruit bats as the natural reservoir of Ebola virus.

Morphology:

The virus undergoes massive replication in endothelial cells, mononuclear phagocytes and hepatocytes. Necrosis is most severe in the liver, spleen, kidney and lymph node. The lungs are hemorrhagic and petechial hemorrhagic are present in skin, mucous membrane and internal organs.

Fungal infections

Fungi are primitive eukaryotic micro-organisms which are now usually classified as neither plants nor animal. Only a few of the very many known species are pathogenic to man, and with some exceptions the lesions are superficial and not serious. A good example of such infection is thrush (candidiasis). However in drug addicts, severely ill patients and particularly in those with T-cell deficiency or on immunosuppression therapy, some fungi can cause more extensive or even systemic infections

Candidiasis

Residing normally in the skin, mouth, gastrointestinal tract, and vagina. *Candida* species usually live as benign commensals and produce no disease. However, *Candida* species, most often *C. albicans*, are the most frequent cause of human fungal infections. These infections range from superficial lesions in healthy persons to disseminated infections in immunocompromised patients. *C. albicans* grows best on warm, moist surfaces and so frequently causes oral thrush, vaginitis, and diaper rash. Diabetics and burn patients are particularly susceptible to superficial candidiasis. *Candida* can be directly introduced into the blood by intravenous lines, catheters, peritoneal dialysis, cardiac surgery, or intravenous

drug abuse. Severe disseminated candidiasis is associated with neutropenia secondary to leukaemia or anticancer therapy and immunosuppression after transplantation.