TRYPANOSOMIASIS

Trypanosomes are flagellated motile protozoan parasites that live in blood and body fluids of their host and localize in tissues, sometimes in a non-flagellated form.

Trypanosomes have certain common features, such as an ovoid or rounded body in the non-flagellate stage and a slender elongate body when it becomes flagellated; a flagellum which arises from the blepharoplast; an undulating membrane which extends along the border of the trypanosome, with the flagellum forming its margin; and the para basal body. A relatively large nucleus is usually located near the middle of the body.

Trypanosomes can usually be identified by their morphological features, such as size, shape, position, arrangement, and development of the organelles.

Specie	Disease	Definitve HOST	Intermediate HOST	Distribution	
Trypanosoma brucei	**Nagana Disease**	Domestic mammals; except Goat	Tsetse fly (*Glossina* Sp.)	African trypanosomiasis Tse-Tse Fly Disease	
Trypanosoma caprae	Trypanomiasia	Horse, sheep, cattle	Tsetse fly		
Trypanosoma congolense	Trypanomiasia	Cattle; Horse; Goar;Sheep; Pig; Dog, Camel	Tsetse fly		
Trypanosoma cruzi	Americal Trypanosomiasia **Chagas Disease**	Man, Dog, Cat etc	"Kissing bug" (Triatoma sp.)		
Trypanosoma equiperdum	**Dourine**	Horse, Ass	Transmitted by **Coitus**		
Trypanosoma evansi	**Surra**	Horse, Ass, Mule; Cattle	**Horse Flies** (Tabanidae and Stomoxys)		
Trypanosoma equinum	**Mal De Caderas** 'Disease of the Hip'	Horses	T equinum is identical to T.evanis, except that it lacks Kinetoplast.		
Trypanosoma gambiense	Chronic Sleeping Sickeness	Man; antelope	Tsetse fly		
Trypanosoma vivax	Trypanomiasia	Cattle; Sheep; Horse; Goat; Camel	Tsetse fly		

LIFE CYCLE:

Almost all trypanosomes are transmitted by arthropods, which act as biological vectors. The only exception is *Trypanosoma equiperdum,* cause of dourine, which is transmitted by coitus. Mechanical transmission of trypanosomes may be done by certain biting flies (Tabanus, Stomoxys), but this is not an important means of spread, except for *T. evansi,* the cause of surra in Camel, Horses, cattle and buffaloes.

Definite cyclic development occurs in the body of true invertebrate hosts. In these hosts, trypanosomes multiply in various forms in the digestive tract, finally migrate as infective forms to the salivary glands and are injected into the mammalian host with the vector's saliva at the time of the bite. *T. cruzi,* the cause of American trypanosomiasis, is an exception. In this case, the infectious stage is located in the hind gut and is excreted in the faeces of the arthropod (Triatoma sp.), when the arthropod defecates at the time of its bite.

When transmitted through vector saliva, trypanosomes are referred to as **Salivarian,** and when through arthropod faeces as **Stercorarian**.

PATHOGENESIS :-

The mechanisms that induce disease or cause death are basically unknown, with the exception of Chagas disease (American trypanosomiasis) caused by *T. cruzi.* This trypanosome invades cells, whereas trypanosomes of the African trypanosomiasis do not. Following entry into the mammalian host, Trypanosomes of African Trypanosomiasis (salivarian) rapidly multiply by binary fission (as Trypanomastigotes) within the bloodstream, leading to parasitaemia.

The parasitaemia remains mostly unaffected and is not diminished by the host's immune response. This is because of the parasite's unique ability to undergo almost endless anti genic variations through changes in surface glycoproteins. Because of their variability, these surface antigens are called variant or variable surface glycoproteins (VSGs). Trypanosomes may also enter the interstitial space and multiply there.

Continued stimulation of the immune system by VSGs explains the reticuloendothelial, Lymphocytic, and plasmacytic (of plasma cells) hyperplasia seen in the spleen and lymph nodes of many affected animals. Glomerulonephritis and vasculitis, which may occur in chronic trypanosomiasis, can also result from the continuous immune response and the formation of antigen-antibody complexes.

Anaemia is a common finding. Although its pathogenesis is not settled, it may also be immunologically mediated. It is in part haemolytic and in part due to erythrophagocytosis.

Just as a drug can adsorb (adhere to the surface) on red cells and make them immunologically foreign, so also the trypanosomes. These altered cells, being regarded as foreign, are either lysed by antibody and haemolytic complement, or are phagocytosed by mononuclear phagocytes. Clinically, severe anaemia is, therefore, characteristic of trypanosomiasis.

The pathogenesis of inflammatory reactions in various tissues is also considered to be immune mediated. These reactions are mainly characterized by proliferation and activation of macrophages. Toxic products of trypanosomes may also play a role in the pathogenesis of tissue damage, which can include necrosis. Usually leukocytosis, thrombocytopaenia, and hypergammaglobulinaemia occur.

SURRA

Surra is an acute, subacute, or chronic disease that **primarily affects Camels & Horses**, but also occurs in cattle, buffaloes. dogs. In India, besides Camel and Cattle, surra is widely prevalent in buffaloes also. It is caused by *Trypanosoma evansi.* Wild ruminants can act as reservoirs. *T. evansi* is transmitted mechanically by the bite of **Horse Flies** (Tabanus, Stomoxys).

The disease is characterized by fever, progressive emaciation, anaemia, subcutaneous Oedema, nervous signs and death. It is regarded as the **most important health problem in camels**. In India, the incidence of surra increases significantly during the rainy season when there are large biting fly populations, the so-called "surra season". Mortality in horses and camels is nearly 100% if untreated, but is much lower in cattle and buffaloes. The survivors become carriers.

The trypanosomes are inoculated into the host from the contaminated mouth parts of the biting insects. **The parasites multiply in the blood and body fluids, including the cerebrospinal fluid, and cause inflammatory changes and anaemia**. Immune mechanisms are related to antigenic variation of the parasite and the production of antibodies by the host.

Clinical Signs

Surra usually occurs in a acute, severe form. The main clinical signs include intermittent fever associated with trypanosomes in the blood, (i) cutaneous plaques and oedema of dependent parts of the body (limbs, lower abdomen and thorax). (ii) gradual emaciation inspite of good appetite. (iii) nasal and ocular discharge, (iv) Progressive Anaemia, (v) delirium and convulsion.

Surra is always fatal in camels and horses, death occurring within a few days or a few months.

However, camels may exhibit chronic signs for years. These signs include a reduction in milk yield and capacity for work, and a high abortion rate in pregnant females.

Cattle and buffaloes in enzootic areas usually have mild infections, which may be aggravated by stress, such as from bad climatic conditions, work, or intercurrent disease. Surra may affect the quality of semen in bulls, and cause irregular oestrus. abortion and stillbirths in cows.

Lesions

Grossly. the carcass is **markedly Emaciated** and pale and may be **Jaundiced**. There may be **patchy Alopecia and Petechiae and Ecchymoses** of visible mucosae. Spleen and Lymph Glands are Enlarged. Congestion of Bone marrow. However. as in *T. brucei* infection, there are no pathognomonic gross or microscopic lesions, although a lymphoplasmacytic (lymphocytes and plasma cells) infiltrate of various organs, including the brain and spinal cord, is characteristic. Trypanosomes can be detected in body fluids.

Diagnosis

- Laboratory help is required to confirm a diagnosis, and even then surra cannot be easily differentiated from *T. brucei* infection where both coexist. Trypanosomes are readily seen in blood smears from animals in the acute phase of the disease. In the chronic phase, repeated sampling for some days may be required, or the trypanosomes in the blood could be concentrated into the buffy coat layer by centrifugation before examination.
- Mercuric Chloride Test: In carrier camels, useful to detect & is 99% accurate. Mix 1 drop of serum with 1 cc of 1 in 25,000 solution of Mercuric perchloride in water. White Precipitate appears in few minutes in positive cases.
- Formol Gel Test: Mix 2 drops of Formaline with 1 cc of serum and keep for 24 hrs. A complete Gelation is Positive. Its only 75% accurate.

Dourine

Dourine is a venereal infection of horses and donkeys caused by *Trypanosoma equiperdum.* Dourine is characterized clinically by inflammation of the external genitalia, cutaneous lesions and paralysis.

In contrast to other trypanosomes, *T. equiperdum* is transmitted by coitus, rarely by biting flies.

The disease is manifested by (i) Oedema of Genital Tract and Ventral Abdominal Oedema, (ii) Urticarial Plaques in the genitalia and skin, (iii) Progressive Emaciation and sometimes by Anaemia. (iv) In CNS, Incoordination and Ascending Motor Paralysis.

T. equiperdum is demonstrable in the lesions, particularly those of the genitalia. The lesions are those of a **Mononuclear Or Granulomatous Inflammation**.

Chagas Disease

Also known as "American trypanosomiasis", Chagas disease is caused by *T. cruzi.*

Chagas disease is transmitted by blood-sucking bugs i.e. "Kissing bug" (Triatoma sp.).

Dogs, cats, pigs, monkeys and small wild animals harbour and also suffer from the disease like humans. *T. cruzi,* in contrast to other trypanosomes, multiplies within the cytoplasm of cells in the mammalian hosts. After release of the infectious stage in the **faeces of the Triatoma bug,** trypanosomes enter the bloodstream, but do not multiply within the bloodstream. Instead, they invade host cells, **particularly cardiac and skeletal muscle**. Here they **transform to amastigotes**, a form closely resembling Leishmania and usually referred to as the leishmanial form of *T. cruzi.* The amastigotes multiply within the cytoplasm of the infected cells, forming collections of organisms called **"pseudocysts".** The amastigotes differentiate into **trypanomastigotes** (Le. typical trypanosomes), which are released upon rupture of the infected cells into the circulation. In the circulation they are available to infect the intermediate host, or invade a nother cell to repeat the cycle.

Lesions

Lesions result from the growth and activity of *T. cruzi* in the blood, but are **influenced by its intracellular activities, particularly in the myocardium**. The initial lesion, in humans, following the bite of a bug is a hard, red, painful oedematous mass at the site of the bite. This soon subsides and the organisms spread. The Lymph nodes become enlarged, oedematous, and sometimes may contain microabscesses.

The heart is particularly affected. Myocardial fibres are penetrated by *T. cruzi.* The trypanosome proliferates fills and destroys muscle fibres, and causes **severe Myocarditis**. The heart becomes enlarged, and the pericardial sac distended with fluid. Large cystic collections of leishmanial forms can be seen microscopically in cardiac muscle cells. Skeletal and smooth muscles m ay also be invaded.

In brain, *T. cruzi* may produce oedema and congestion, particularly in the Meninges. Testicle may be severely invaded by *T. cruzi.*