REVIEW
OF
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TORCH :- Consist of

Toxoplasma gondii.
Rubella virus.
Cytomegalovirus.
Herpes simplex virus.

Toxoplasmosis :- It is a common central nervous system infection in patients of HIV disease.

EPIDEMIOLOGY :- Toxoplasma gondii is an oblique intracellular parasite with worldwide distribution found both in man & animal caused by a small protozoan. Approximately 3300 infants born every year in the United States are congenitally infected with Toxoplasmosis, most infants are asymptomatic during neonatal period but subsequently develop adverse sequelae. Early treatment reduces the severity of the disease.

Development forms includes the oocyst, trophozoites tissues cyst. Organism reproduce sexually in the intestinal mucosa of cats, the only definative host to form oocysts, which are excreted in the stool., oocysts becomes infectious when they undergo sporogony outside the body. Humans are infected following ingestion of oocysts from dried out faeces on tissue cyst from contaminated food especially under-cooked meat. High rates are associated with tropical climates, poor sanitary condition, a prevalence of cats.

PATHOGENESIS :- Following the ingestion of T. gondii trophozoites disseminates throughout the body via the
lymphatics & blood strom, organisms invade cells and multiply resulting in tissue distractions cyst containing several thousand slowly growing organisms may develop in any tissue but are common in CVS, Myocardium & skeletal muscles, HIV induced immuno-suppression permits reactivation - dissemination of latent infection.

**CLINICAL FEATURES** :- Four main type of clinical illness has been described.

(1) Acquired:

(A) **Cerebrospinal**: Common in children presents as acute meningo-encephalitis.

(B) **Lymphatic**: Presents as lymphadinitis of one or more group, fever lasting for several weeks with constitutional symptoms.

(C) **Exanthematous**: Mostly present in adults characterised by acutes febrile illness with wide spread maculo papular rashes, diffuse interstitial pneumonia, Myocarditis occasionally meningo-encephalitis

(D) **Latent**: Does not produce any sign or symptoms and infection is detected by laboratory tests, it is found only in adults.

(2) **Congenital**: It may produce obstructed labour due to hydrocephalous; congenital infections are usually fatal, hydrocephalous develops in majority of infants, extensive cerebral lesions gets calcified & if child survives leads to Jacksonian epilepsy, children are mentally deficient.
**DIAGNOSIS:** Definitive diagnosis of Toxoplasma requires demonstration of free or intracellular trophozoites in tissue, nucleated cell, or body fluids.

Primary infection with T. Gondii in the normal host generally results in seroconversion. Antibody is measured by the Sabin feldman exclusion test or by ELISA test.

Leucocytosis with lymphocytosis is present. CSF examination shows xanthochromia with raised proteins normal sugar and lymphocytes.

**TREATMENT:** A combination of pyrimethamine 25-50 mg three to four times a day and sulphadiazine initially 2g and then 1g every 6 hourly for 14 days is given in all cases.

In primary, Azithromycin 500g 1BD for 15days Spiramycin 6-9 MIU 1 day in devided for 15 days

Kanta and I.al et al (1990). Toxoplasmosis is the epidemic infection by the parasite called (Toxoplasma gondii) foetal infection is via placenta during parasitamic phase and infection during early pregnancy is most damaging significant corelation of rasied tetre of Toxoplasma antibody with bad abstetric history i.e. abortion, premature labour, still births, neonatal deaths and congenital abnomalities.

Toxoplasmosis is of world wide distribution. It is a multysystem disease of great hazard to the foetus. It is caused by hand to mouth contact after the handling of infected cat faeces, or infection of meat from infected cattle or sheep. Infection in the first trimester can lead to abortion and
congenital abnormalities. It does not affect subsequent pregnancies except in the event of reinfection (Sofia Rajan et al (1986-1988).

Risk of infection - (Couvreur 1979) pregnant women who have been infected before conception are immune because they have developed specific toxoplasma antibodies in their blood (seropositive) those who have not been infected lack these antibodies (seronegative) and susceptible to toxoplasma infection.

The rate of infection increased with gestational age from 14% when infection was acquired in the first trimester to 29% in the second and 59% in the third trimester.

Sauchitra Dashora et al (1988) studied 8 cases with 3 or more pregnancy losses and 20 cases with good obstetric history as control. She studied the group which comprised of 38, 24, 27, and 7 cases having abortion, preterm births, stillbirth and neonatal deaths respectively.

The incidence of toxoplasmosis in one case was found to be 9% significant seropositivity was found only in 5 cases in study group. Positive history of contact with cats and dogs was present in 15% cases of study group only. Seropositive patients showed a higher incidence of abortion, preterm birth and stillbirth.

Malathy Kuppuswamy et al (1988) a case report Mrs K 21 year old in the Jan 1986. She had lost two children, She had been married for 6 years. The first, a male preterm (8 months) home delivery baby had a maculopapular rash all over the body at the time of birth. The rash showed acute
exacerbation and remission and was seen to regress over one part of the body and reappear over other. There was no rash free period during the one year that the child lived. The child had no abnormalities and had normal milestones. Immunization not carried out. The second delivery was fullterm male child average birth weight, the child had similar skin lesions as in the first child and a ‘small head’. This baby died after 20 days. Both children were restless and suffered loss of appetite 2 weeks prior to death.

Eiji Konishi et al (1989) obtained 600 pregnant women during the first trimester and from 1200 umbilical cord of newborns, including 393 paired sera. The change of IgG antibody level indicated on increase of $> 0.1$ in 11 pairs (2.8%) a decrease of $> 0.1$ % in 6 pairs (1.5%) and no significant change in 376 pairs (95.7%) increase was observed in sample with relatively low initial level and vice-versa. 3 pairs increased from $<$ to 0.1, to $> 0.1$ which suggest newly acquired infection during pregnancy, but thier increasing level were $< 0.3$ in 11 pairs showed an increase in IgG antibody level, all infants were normal without any clinical sign suggestive of congenital Toxoplasmosis.

Tomba Singh et al (1992). HE took 120 patients of high risk pregnancies and 100 normal pregnancies were studied incidence of maternal Toxoplasmosis was 13.3 % in high risk pregnancy while it was 4 % in normal control group.

Kusuma sexena et al (1993) studied 150 cases 50 control and 100 test group of had obstetric history was 27%. Maximum positive cases were found in age group of 21-30 years, almost equal incidence of positive cases in rural &
urban population. Positive cases were more in non vegetarian and in patients who has history of contact with cats. The positivity was 26.2% in abortion cases 30.7% in cases with history of premature labour, 25% cases with history of still birth and 33.3% in cases with history of congenital malformation.

Abortion - 61 cases 16 (26.2%) were positive.
Premature Labour - 13 cases 4 (30.7%) were positive
Still birth - 20 cases 5 (25%) were positive
Congenital malformation - 6 cases 2 (33.3%) positive

Hohlfeld et al (1994) studied 2632 women between 25 and 36 weeks of gestation with gondii infection IgM positive rate was 12% among infected foetuses in which blood sample obtained for 25 weeks gestation, compared with 31% between 25 and 30 weeks and 5.9% after 30 weeks. The overall fetal risk was 7.4% but varied with gestation age.
RUBELLA

Historical aspect of Rubella:

Clinical Rubella (Three day measles or German Measles) was first discovered in Germany about 200 years ago and was called Rothelin. In the late 1930’s the Viral agent was transmitted to man and monkeys, but was considered an inconsequential disease until 1941 when Gregg an Australian ophthalmologist discovered the teratogenic property of rubella. He observed a sudden increase in congenital contract in infants and related it to maternal rubella.

Rubella virus was isolated in tissue culture in 1962 independently by two group of worker (Weller and Neva. Parkman, Bueschan and Arkenstien) in 1969 the first rubella vaccine was licenced in United states.

Thus in a relatively short time cause of neonatal rubella was identified. pathogenesis explained and a preventive measures created and then public health measures reduced the incidence of disease.

Structure and properties of Rubella

Rubella is a RNA virus of Togavirus group contain 11camagglutinins. There are several stain of the virus, the virus is readily inactivated by that heat and chemical agents. The viral particle is roughly spherical 50-70 nm. in diameter surround by envelope. It is inactivated by ether chloroform.
and formaldehyde. It is destroyed by heating at 56°C but survives for several years at 60°C.

Clinical infection by Rubella virus during pregnancy:

Infection takes place though the nasopharynx after that virus incubates in the lymph nodes for 15-21 days (20 days incubation period), which is followed by prodromal viraemia marked by coryza, malaise and low grade fever.

This is followed by discrete maculopapular rash appearing first on face than spreading to neck, trunk disappear by 3rd day.

However about half to two-third of cases of Maternal rubella are clinically inapparent.

Immune phenomenon in the body:

Anti-Rubella IgG antibody of Maternal origin are transferred transplacentally to the developing foetus. In event of en-utero infection immune response in the form of rubella specific IgG antibodies is seen for a duration of two months in the foetus. It may last upto an year. This is followed by IgG antibodies response which can last for around 10 years. Maternal IgG transfer across the placenta generally last for 6 months. In general it can be said that cases negative for antibodies are always at risk and cases positive for antibodies remains protected for a minimum of 10 years (Khare et al 1987, Bhaskaran et al 1991)
Diagnosis of Rubella

The other store for the diagnosis of maternal rubella infection is serologic using.

Most widely used test being.

Heamagglutination inhibition Test. It is time consuming and complex and is being replaced by newer methods namely.

Solid phase enzyme linked immunosorbent assay (ELISA)

Passive heamagglutination (PHA)
Immunofluorescent assay (IFA)
Radial immunodifussion test (RID)

Demonstration of rubella antibodies by these newer techniques constitute a proof of immunity and demonstration of seroconversion implies recent infection. Recent infection can also be rubella IgG antibodies in maternal serum. Foetus infection can diagnosed by detection of rubella specific IgG in foetal blood obtained by cordocentesis or foetoscopy after 22 wks of gestation.

Contribution of rubella bad obstetric:-

Maternal rubella in the first trimester of pregnancy is likely to result in birth of a malformed baby or induction of spontaneous abortion. Such sequelae of congenital rubella as gross cardial lesions, hepatitis, Meningoencephalitis and interstitial pneumonia are often fatal to infants other such as pancytopenia, retinopathy, Hepatosplenomegaly,
Lymphadenopathy and bone lesions are usually self limited and seems to pose no long term risks. In adult who have survived congenital rubella major handicap with psychomotor, perceptual and cardiac abnormalities as less common complication.

The original concept of congenital rubella syndrome consisting of the triad of contract congenital heart disease and deafness in the new born is no longer accepted as the only evidence of rubella infection (Marshal 1973).

Criteria for Diagnosis of Congenital Rubella Syndrome:-

Have been laid down by centres of disease control, Atlanta, Georgia, in two categories. These are:-

1. A confirmed case which shows a congenital defect typical of congenital rubella syndrome. This can be followed by either isolation of rubella virus or rubella specific IgM antibodies and a persistently high HIA.

2. A compatible case which has sufficient laboratory data, associated with two of the congenital malformations cataract, glaucoma, patent ductus arteriosus, deafness or lymphadenopathy. Some of the cases showing malformation.

Prevalence Studies done on Rubella :- The epidemiological survey have shown a world wide prevalence of rubella infection including South East Asia. Approximately 20% women in child bearing age do not show immunity to rubella. Similar result have been reported by
(Seth et al 1971 and Pal et al 1974) from the Urban population of New Delhi and Chandigarh respectively. However the rural population around Delhi had a much lower immunity (Seth et al 1971) an from Urban Calcutta, Chakraborty et al 1973 also reported a much lower immunity (54%). These data suggest that a sizeable population of India is susceptible to infection by rubella. A number of studies have demonstrated the universal presence of rubella and its predominance in women and children (S. K. Khare 1987). In a study of 160 pregnant women at National institute of Communicable disease, Delhi has shown that 50% of those women were seropositive for rubella.

Serum titre for antirubella antibodies were measured in 2362 female patients of childbearing age group in a large city of Southern Italy by micro haemagglutination inhibition technique and sera were screened for IgM antibody by ELISA (Lecourand G. Et al 1993).

**Rubella vaccination**- In 1969 two vaccines were licenced in United States, the HPV 77- DES and cenderhill strain. HPV 77 was most commonly used vaccine until it is replaced by RA 27 / 3 in 1979. RA 27/3 mimics natural rubella better and more consistently than other vaccines. It is a live attenuated vaccine and is contraindicated in pregnancy. However, the risk of foetal infection after vaccination is between 3% to 5%. Vaccine produces seroconversion in 95 to 98% of susceptible individuals and causes symptoms resembling mild rubella in 10% to 15% of recipient.
CYTOMEGALO VIRUS INFECTION

Cytomegalovirus is a ubiquitous D.N.A. virus belonging to the family of human herpes virus, that includes herpes simplex virus, Ebstein barr virus, varicellazoster virus. The virus ranges in size form 180 to 250 nm in diameter and has a genome in the range of 150 million daltons.

Epidemiology- Most people are infected with CMV sometime during their life. Antibodies to CMV have been detected in over 90% of homosexual men and 60% of women attending sexually transmitted disease clinics in the United States. Cytomegalovirus can be cultured from saliva, semen, vaginal secretions blood or infected tissues.

Common modes of transmission includes sexual intercourse, sharing contaminated needles blood transfusion and perinatal transmission vertical transmission may occur at delivery through contact with infected secretion or post partum via contaminated breast milk . Infected organs used for transplantaion are also a potential source of CMV infection. Infection may be subclinical or clinical later may result in.
* Generalised disease without localising features.
* Hepatitis with pyrexia.
* Glandular fever like illness with negative paul bunnel test.
* Post perfusion syndrome.

A primary maternal infection in any trimester has the potential for congenital transmission. Studies by Monif et al and Stern and Tucker have shown that maternal infection in any trimester can lead to foetal infection . More severely
affected infants were born to mother who developed infection during the second rather than during the third trimester. The risk of intrauterine infection following primary CMV infection in pregnancy 30-40% as determined by neonatal urine cultures.

CMV infection can be acquired at the time of vaginal delivery. Another common route of infection is breast feeding. Virus is transmitted in the breast milk of 25.7% of women with serologic evidence of CMV infection.

Other potential route but rare source of infection is introduction of virus into foetal blood stream or amniotic fluid during intrauterine transfusion or amniocentesis.

There is variation in the frequency and severity of the congenital infection depending on the nature of the maternal infection when infection is primary 30-40% of the neonates will be infected and 10% of them will exhibit overt CMV infection. The risk is less when infection is recurrent and only 2-3% of the baby will be infected.

Severe congenital infection - Infants born with severe congenital infection exhibits hepatosplenomegaly, thrombocytopenia with petechiae & purpura, hepatitis associated with icterus pneumonitis and chorioretinitis, abnormality that results from faulty neurologic development includes microcephaly, optic atrophy, Aplasia of various parts of brain and microphthalmia. Incidence of foetal growth retardation is 30-40%. The presence of intracranial calcification is an indication that the infants will have at least moderate to severe retardation. Mental retardation affects 95% of new borns with severe infection, Auditory deficiency
is the most common handicap affecting 25% of congenitally infected new borns.

**Diagnosis:-**

**(1) Culture-**
The gold standard for the diagnosis of CMV infection is the viral culture. Virus can be cultured by conventional technique or by the shell viral methods. Culture for CMV is expensive and virus may require up to three weeks to grow.

**(2) Histology-**
Histologic examination of biopsy specimens may demonstrate typical CMV inclusions, tissue necrosis or both. Definative histologic diagnosis of CMV infection requires the presence of characteristic ‘OWL.’s eye cells with cytomegaly and large intranuclear or interacytoplasmic inclusions surrounded by a halo. Immunofluorescent staining of frozen section using an anti CMV monoclonal antibody may facilitate diagnosis and increase the sensitivity of cytologic examination.

**(3) Serology-**
Serologic tests are useful in the diagnosis of acute infection characterised by IgM antibodies recurrent infection is characterised by at least four fold increase in IgM titres. Antibodies may be absent in AIDS patients with severe CMV infection and a negative titre does not excludes the possibility of active disease there fore serologic testing for CMV is of little diagnostic utility in HIV infected patients.
(4) Treatment-

Cytomegaloviral infection is neither preventable nor curable. Efforts are being made to produce a vaccine. Antiviral agent gancyclovin is a potent inhibitor of CMV replication. The medication is available only for intravenous administration and have significant toxicity.

Nage (1967) said the gestational age of affected children varied from 20-40 weeks with a mean of 36.2±3.4 weeks. The baby wt. as percentage of expected gestational baby weight was 82±17%. All babies were below expected weight and were slightly premature except two.

Stagnac described congenital CMV infection in two siblings born 3 years apart the older child had viraemia at birth severe growth retardation hepatosplenomegaly and later severe psychomotor retardation. The second infants had virumia at birth but remained mostly asymptomatic.

Hanshaw et al (1976) reported that 5 of 40 (13%) infants with asymptomatic congenital CMV infection had bilateral hearing loss and 3 had deafness.

Reynolds et al (1974) suggested that some of the estimated 1 in 1000 cases of unexplained profound deafness in American children may be caused by congenital CMV infection.

Lawrence Hatherley (1985) said 12 neonates in 47, 320 consecutive births cytomegalic inclusion disease confirmed by viral studies. an incidence of approximately 1 in 4000 deliveries, further 4 cases were diagnosed in 738 (1 in 185) neonates admitted for intensive care, congenital CID was
diagnosed in 12 to 16 neonates and post natal infection in remaining 4 infants, the sexes were equally represented which included 1 set of twin. Five death occurred in hospital between 6 hours and 135 days of delivery. 11 infants were discharged for followup and 7 showed CMV infection.

Confirmed by
Blood
Urine
Saliva
Placenta
Cerebrospinal fluid
post mortum specimen

Sterio Stago et al (1991) studied 132 cases resulting from primary maternal infection and 63 cases which had recurrent maternal infection. Primary maternal infection was defined by seroconversion and the detection of CMV specific IgM antibodies during gestation. Recurrent maternal infection was defined by the presence of IgM specific antibodies at least 9 month prior to conception, or CMV positivity with in the first 12 weeks of gestation but without CMV specific IgM antibodies during this time.

Griffiths and Babrobin (1992) pointed that fetal loss occurred in 4/25 (5%) of early CMV infection. The transmission of CMV from mother to foetus in early pregnancy is very high.

Transmission of CMV = Transmission of HIV-1 in 8 weeks foetus.
They are suggested that potential CMV carrier may transmit CMV to their foetus in early pregnancy. The rate of CMV IgM is 5.6% but the incidence of congenital CMV infection of chorionic villi in early pregnancy was 23.5%.

Chiba (1992) said congenital CMV infection after secondary maternal infection, common in Japan. Did not describe the route of infections whether hematogenous or ascending or in the birth canal (Chibe, et al 1992 Ahlfors et al 1993).

In patients, post transfusion infection could be excluded, since the infant was positive for anti CMV IgM before receiving transfusion, the breast milk was doubtful source of infection since the period of incubation is too short for seroconversion. Transmission through birth canal is unlikely by caesarean delivery CMV had ascended from vagina via ruptured membrane to reach the decidua or amniotic fluid, the foetus than aspirated the virus to develop a congenital pneumonia.
HERPES SIMPLEX VIRUS

Herpes simplex virus is of major obstetric interest because it is one of the most common sexually transmitted disease and because of its potential to cause severe foetal and neonatal infection.

Epidemiology: HSV belongs to the herpes virus family who have the ability to persist throughout the life of their host to produce recurrent infection. Primary infection occurs 2-12 days after the introduction of the infectious secretions into the oral cavity (HSV-1) genital area (HSV-2) skin or eyes 50% of adults have antibodies to HSV with a higher prevalence in certain populations including male homosexual and those from developing countries.

Clinical Features: -

In Infants: 1. Acute gingivostomatitis, common age of occurrence is of 10 months to 3 years, onset is abrupt with fever, generalized malaise, irritability and soreness of the mouth lesions are seen just inside the lips. After healing mucosal lesions do not reoccur but skin lesions may reappear over a period of many years, these vesicles are called cold sores, common precipitating cause of recurrence is febrile illness, trauma, exposure to sunlight.

2. Genital Herpes: -

Vulvovaginitis: In female infants HSV may be the cause of vulvovaginitis typically there are herpetic eruption
around the vulva area becomes inflammed and painful , in males there may be herpes of glans penis .

3. **Infection of the eye :-** The most common herpetic infection of the eye in infants is acute oedematous conjunctivitis with chemosis and stickness of the lids . The preauricular lymphnodes are enlarge and tender . Corneal involvement lead to formation of dendritic ulcer and loss of vision.

4. **Skin Infection:--** Eruption involves any area of the skin common are napkin area of the skin.

5. **Meningitis :-** It may be the rare cause of meningitis.

6. **Acute generalised infection :-** Neonatal infection with HSV is often life threatening and causes severe morbidity amongst some infants.

Genital herpes simplex in pregnancy poses a risk for transmission of infection to the foetus at birth ,during prolong rupture of membrane as well as possibly a risk for congenital malformation of the unborn . In late 1960 it was discovered that there are a type of herpes simplex virus infecting humans . Type -I causes a majority of oral infection and Type -II causes genital infection with either type is often asymptomatic but often gives a long lasting IgG response . Neonatal infection with HSV is often life threatening and causes severe morbidity among some infants ( Whitley et al 1980 Stone et al 1988) . Genital herpes in pregnancy are associated with increasing foetal and maternal morbidity and mortality ( Nahmians et al 1971 ) . HSV has been reported to increase spontaneous abortion maturity in
uterodeath and neonatal death in this study comparing the pregnancy outcome of 15 patients with primary genital herpes and 14 with non-primary 1st episode disease complicating pregnancy. 6 of the 15 (40%) patients with primary infection developed a serious obstetrical and perinatal complication.

Adverse outcome increased with advancing gestation with 1 of the 5 cases in 1st trimester, 1 of the 5 cases in 2nd trimester and 4 of the 5 cases in 3rd trimester demonstrating 1 or more of these complications.

John 1988 suggested that neonatal herpes infection occurs in about 1 in 500 pregnancies in U.K. but rare in Britain. Between 1976 and 1985, 6 million births were recorded in England and Wales but only 80 neonatal deaths were reported from herpes. Herpes infection of the newborn is thus much less common than infection with genital herpes simplex virus in women of reproductive age. The transmission of herpes infection from baby is high (40%) in primary maternal infection but low (3%) in recurrent maternal infection.
HUMAN IMMUNO DEFFICIENCY VIRUS (HIV)

Epidemiology:

The human immuno deficiency virus (HIV) is the cause of acquired immuno deficiency syandrome (AIDS) a condition that affect thousands of individuals in united state and many through out the world. The demographies of this disease are changing and HIV is infecting a growing number of women of reproductive age. As a consequence, the number of infants born to HIV infected mother is also rapidly increasing.

1. Virology

A. Causative Agent: In 1983 -84 the virus responsible for AIDS was identified by young french researcher Francoise Barre Sinoussi, working in montaingeners laboratory with the lymph node of a patient with early AIDS. It was called lymphadenopathy associated virus (LAV) research in USA called it Human T cell Lymphotrophic virus III (HTLV III), In May 1986 the international Committee of the Taxonomy gave it a new name Human Immuno Defecency Virus (HIV) It belongs to the family of Lantiviridae of retrovirus group, which includes Human T cell Leukemia virus -1 (HTLV-1), It contains a unique enzyme, Reverse transcriptase (RT).

The virus is 1/10000 th of a Millimeter in diameter. It is a RNA virus, has a protien capsule containing two short stands of genetic material (RNA) ands few enzymés.
The surface of virus has envelope has 72 uniformly arranged glycoprotein knobs(gp) the envelope glycoprotein are designated gp 160 , gp 120 and gp 41 of the gp 120 contains the binding site for host receptor cell . All cells of the body possessing the CD 4 + receptor site , the special binding site for gp 120 of the virus , are susceptible to HIV . Other viral infections eg . Cytomegalo , Virus , herpes virus etc. can make more susceptible cells prone to HIV infection by acting as cofactors CD4 + lymphocytes are the major target cells . B lymphocytes , Macrophages , Megakaryocytes , Microglial cell macrophages in brains , fibroblast and langerhans cells in skin and mucosal cells of the bowel etc. have been found to get infected . Thus HIV infection is polytropic rather than solely lymphotropic , but its level of replication in different cells varies .

Farthing et al ( 1986 ) described the replication of viruses . Human retroviruses are subdivided into two categories .

1. TRANSFORMING RETROVIRUSES :-

Includes HTLV-I and HTLV-II . They are monocytolytic and caused neoplastic transformation of its target cells , giving rise to a T cell leukemia .

2. CYTOPATHIC RETROVIRUSES :-

Includes HIV-I and HIV-II , these are cytolytic for T cells and hence profound immuno deficiency rather than primary neoplasia based on comparisons of morphology , genomic structure , antigen reactivity , and biologic behavior. They are more likely relative to family of animal retroviruses.
The virus is easily killed by heat, it is easily inactivated by ether, acetone, ethanol (20%) and beta propane action (1:400 dilution) but is relatively resistant to lowering radiation and ultraviolet light (WHO 1994).

B. Reservoir of infection:

There are cases and carries once a person is infected, the viruses remains in the body life long. The risk of developing AIDS increases with time since HIV infection can take years to manifest itself, the symptom carrier can infect other people for years.

C. Source of Infection:-

The viruses has been found in greatest concentration in blood, semen and CSF, lower concentration have been detected in tears, saliva, breast milk, Urine, cervical and Vaginal discharge. HIV has also been isolated in brain tissue, lymph nodes, bone marrow, cells and skin.

2. HOST FACTORS: -

(A) AGE: - Most cases have occurred among sexually active persons 20-49 years. This group represents the most productive member of the society and those responsible for child bearing children under the age of 15 makeup less than 3 percent of cases.

(B) SEX: - In North America, Europe and Australia about 70% of cases are homosexual and bisexual men. In Africa the sex ratio is equal certain sexual practices increases the
risk of infection more than the others eg. Multiple sexual partners, anal intercourse, and male homosexuality, higher rate of HIV is found in prostitutes.

(C) IMMUNITY: The immune system disorders associated with HIV infection, AIDS is considered to occur primarily from the gradual depletion in a specialized group of white blood cells (Lymphocytes) called helper T cells or T4 Cells.

Full name of T4 cells is CD4+ T Lymphocyte. It is also commonly known as CD4+ cells. These cells play a role in regulating the immune response.

HIV selectively infects T helper cells apart from several other cells in the immune system such as B cells, Macrophages and nerve cells. When viruses reproduce the infected T helper cells are destroyed. Consequently people with AIDS tends to have low over all white cell count (Senegal 1991) whereas healthy individual have twice as many helper cells as suppressor cells. In AIDS patients the ratio is reversed. A decreased ratio of helper to suppressor cells may be an indirect indicator of reduced cellular immunity. One of the most striking feature of the immune system of patient with AIDS is profound lymphopenia with a total lymphocyte count often below 500 cu mm. It is the alternation in T cells function that is responsible for the development of neoplasms, the inability to mount a delayed type of hypersensitivity response. The lack of an obvious immunological response by the host to the virus is one of the problems confirming scientist (WHO 1985). That is those with antibodies and these antibodies to HIV and these antibodies are also ineffective against the virus.
**Mode of Transmission:** Basic mode of transmission are:

(a) **Sexual transmission**

(b) **Blood contact**

(c) **Mother to foetal transmission** :- HIV may passes from mother to her foetus through the placenta or to her infant during delivery or by breast feeding. About one third of the children of HIV positive mothers get infected through this route.

The risk of transmission of infection is higher if the mother is newly infected or if she has already developed AIDS.

**Natural History of HIV Illness :-** (Maviya AN 1990)

**Incubation period** :- Incubation period of HIV virus is uncertain (from few months to 10 yrs) from HIV infection to the development of AIDS the virus can lie silent in the body for many years. the percentage of people infected with HIV who will develop clinical disease in uncertain possibly 10-30% will develop AIDS and another 25-30% develop AIDS related complex. However it is estimated that 75% those infected with HIV will develop AIDS by the end of ten years (Sehgal 1991).

**1. Acute seroconversion illness :-** In about 15% of cases HIV infection presents as acute viral illness developing about 6 weeks after the infectious mononucleosis with high fever, skin rash, headache, muscle pains, joint pain and enlarged lymph nodes.

**2. Chronic stage :-** Most of the patients who survive the acute stage of HIV infection enter the chronic stage. During the chronic stage, the body continues to produce antibodies against HIV, but the immune system is weakened and more susceptible to other infections. The patient may experience various symptoms such as fatigue, fever, weight loss, and night sweats.

**3. Late stage :-** In the late stage of AIDS, the immune system is severely damaged, and the body is unable to fight off infections and diseases. Symptoms of the late stage of AIDS include fatigue, fever, weight loss, night sweats, and opportunistic infections.

**4. Death :-** In the final stage, the body is unable to fight off infections, and the patient will usually die within a few months.
lymph nodes in the neck and axilla (Buchanan et al 1986) (Gaines et al 1988) Encephalitis and aseptic meningitis may occur by 2 weeks illness clears up if tested for HIV the person would show a positive serological test during the recovery phase and hence the name “Seroconversion illness”.

(Biggs et al 1986) during this stage these are an attempt on the part of the body to prevent the invasion of the virus by mounting an immune response. The immune system produces substance called antibody in order to check the spread of the virus.

Till this period is very difficult to detect the virus by the commonly used laboratory tests. Therefore it is also termed as “window period”.

2. **Asymptomatic carrier stage**

After infection patients remains healthy with no signs and symptoms of illness (7-9 years) however being a virus carrier he/she can infect his/her close contacts through unprotected sex, infected blood, needle sharing in cases of drug addiction, or accidental injuries some patients pass through a stage of generalised swelling of the lymph nodes. the glands exceed 1 cm in diameter except for this defensive reaction to the presence of HIV infection, there are no other symptoms. This stage called persistent generalised lymphadenopathy (PGL) is to be differentiated from other similar condition and needs to be further confirmed by the ELISA on any other confirmatory test.

3. **Early Clinical Stage** :- During this period the infected person shows clinical signs of the disease these usually
manifest themselves within some years of acquiring the infection and can only be suspected certain innocuous viral bacterial, parasitic and fungal infection which are usually self-limiting or easily treatable in humans appear in a virulent form due to knocking down of the immune mechanism they become life threatening.

In developing country like India, with poor nutrition and a high incidence of infection disease many other causes of these signs and symptoms have to be first exluded. For instance cold sores due to the herpes viruses which appear at the angle of mouth are very common and disappear with out treatment. But in the presence of HIV infection they may become large and cause nonhealing raw areas.

4. Late Clinical Stage: This is the stage of full blown AIDS within a few months of AIDS related complex the immune system of HIV infected person shows further deterioration of helper (CD4) cells fall below 400 cemm in blood and CD4 to CD8 ratio is reversed than these patients starts severe opportunistic infection by wide variety ofd organisms. The resistance of the individual is so poor that many infective againts which do not harm normal persons cause life threatening diseases by taking advantage of the weakend defenses of the body. Hence these infections are called opportunistic infections, increased incidence of serious disease eg. Tuberculosis in India will increase further and in the presence of HIV infection it will become a multiorgan disease causing wide spread damage resulting in high mortality.

Severe opportunistic infection are:-
(a) Fungal infection cryptococcus, candida, histoplasma.
(b) Protozoal infection pneumocystic carinii cryptosporidium isospora, Toxoplasma.

c) Bacterial infection - Typical and atypical mycobacterial infection, salmonella, shigella.

d) Helminthic infections - Stronguloidosis, opportunistic cancers are -

(I) Generalised and aggressive form of kaposi sarcoma.
(II) High grade B cell lymphoma of brain.

c) Central Nervous system HIV disease - During the terminal stage of illness CNS gets involved -

    (I ) AIDS dementia complex - It consist cognitive behavioural and motor abnormalities consisting clumsiness and slowing down of movements disturbance of thought, memory and Judgement.

    (II) AIDS myelopathy - Special type of spinal cord damage (Vacuolar Myelopathy) with a form of sensory motor paralysis, resembling subacute combined degeneration.

    (III) AIDS Neuropathy - It causes severe pins & needles on the tips of fingers and toes. (Roman et al 1988)

**CLINICAL SYNDROME OF AIDS:**

The clinical presentation of AIDS can be catagorised in certain well defined patterns given below.

1. **Pulmonary Presentation** - In the form of
( I ) Tuberculosis ( Atypical )
( II ) Pneumocystis carinii Pneumonia

( I ) Tuberculosis :- Petcheuik et al 1984
Lamouneux et al 1987
Pape et al 1989
Chaission et al 1989

Harries et al 1990 in their successive studies described mycobacterium tuberculosis as a more common pulmonary infection in AIDS patients especially in developing countries. Tuberculosis in AIDS usually involves lower zone of lung cavity formation may not be seen. Fever is prominent and sputum may show acid fast bacilli.

( II ) Pneumocystis carinii Pneumonia - Bigey et al 1986 described the usefulness of sputum inclusion technique is the diagnosis of PCP in AIDS patients with subacute onset of

(a) Progressive dyspnoea
(b) Fever
(c) Non productive cough
(d) Severe respiratory distress with cyanosis ( Sulfxidine et al 1987 )

2. Gastrointestinal Presentation :-

Malebranche et al 1983 described severe gastrointestinal symptoms like unexplained diarhoea, fever, Weight loss, Anorexia, Ophthalmic candidiasis as primary clinical features of AIDS patients.

Dehovits et al 1986, identified isospora and cryptosporidium in over 50-60% AIDS patients in America
as a causative factor for diarrhoea Quinn et al 1987 described various gastrointestinal manifestations of HIV.

3. Dermatological Presentation :-

Impetigo, pruritis, Chronic folliculitis, Seborrhoic dermatitis, fungal infections, Moluscum contagiosum, warts psoriasis, Herpes simplex, Varicella zoster, purpura are of some well described lesions.

Kaposi's Sarcoma :- friedman Kin et al 1982 first described Kaposi's sarcoma in homosexual man. It appears a red purple flat or raised lesion anywhere on the skin in disseminated form it involves the lymph nodes, Oral Mucosa and visceral Mucosa. GIT being commonly involved causing bleeding and obstruction. Kaposi's sarcoma has not yet been reported in India.

(4) CNS Presentation:-

Fisch et al 1985 and Bertoliatl 1988; reported Toxoplasma gondii infection of CNS in the form of space occupying lesion cytomegalovirus as a cause of meningoencephalitis and papova virus and helps simplex virus as opportunistic infection causing encephalopathy manifest in the form of AIDS myelopathy and neuropathy (Roman 1987 and Roman 1988)

Diagnosis of AIDS-

Existence of at least two major signs associated with at least one minor sign in the absence of other known cause of
immuno suppression such as cancer severe Malnutrition or other recognised actiology(WHO 1967)

**Major signs-**
(a) Fever for more than one month.
(b) Weight loss more than 10% of body weight.
(c) Diarrhoea for more than one month.

**Minor signs-**
(a) Cough for more than one month.
(b) Generalised puritic dermatitis.
(c) Recurrent herpes zoster on shingles.
(d) Oropharyngeal candidasis or thrush.
(e) Chronic or aggressive ulcerative herpes simplex
(f) Persistent generalised lymphadenopathy.

**Risk factors for HIV infection-**

(A) Present or past high risk behaviour.
(a) Unprotected penetrative and or vaginal sex with several sex partners.
(b) Drug injecting with shared syringes and needles.
(c) Sex partner of some one with a known risk factor.
(d) Sex partner with known AIDS or HIV infection.
(e) Recent history of an STD particularly genital ulcer disease.
(f) History of unscreened blood or other transfusion after 1975 or from an area with high prevalence of HIV infection even if unscreened.
(f) History of sacrifice, tattooing, ear piercing or circumcision using non sterile instruments.

**Control of AIDS.**
There are basic approaches to control AIDS
(1) Education
(2) Prevention of blood borne HIV transmission
(3) Primary health care.

EPIDEMIOLOGICAL ASPECTS:

Global distribution of HIV :-

The epidemiological of AIDS in developed countries in Western Europe, North America has recently been recieved by Alder 1988.

WHO estimates that by 1993 more than 2.5 Million adult full blown cases may have occured world wide, Although AIDS was first recognized in USA in 1981, earlier case were found by retrospective analysis to have occured in 1978 in the USA and in the late 1970’s in equatorial Africa (WHO 1986).

The number of AIDS cases gives a forecase rather than a true reflection with a virus that takes many years to causes illness.

An explosion of HIV has recently occured in South-East Asia, particularly in Thailand, Burma and India where with in only a few years over two Million people may have already been infected,during the next decade HIV is likely to reach most of the communities around the world and geographic boundries cannot protect against HIV .
HIV infection in India

The first group of seropositive individual in India detected in April 1986 were ten prostitutes, with in short span of 18 months it become obvious that the seropositivity rate was low 4/1000 and heterosexual promiscuity was the major mode of transmission in India (ICMR).

As the contraceptive use is quite low and birth rate continous to be high in India, it is therefore not surprising that pregnancies among seropositive women were reported in 1986 itself, first seropositive pregnant mother was detected in Sept. 1986 (ICMR). First seropositive infant was detected in 1987.

Till Nov-Dec. 1990 out of 44 Indian AIDS patients reported to ICMR, 8 were women.

No patients of paediatric AIDS was reported till Dec. 1990.

1st Dec. is being observed as world AIDS day since 1988 1990-91 being observed as “women and AIDS” with following aims.

To continue to increase awareness about HIV/AIDS.

To strengthen the world wide efforts to stop AIDS by highlighting the impact of HIV/AIDS in women around the world.
To support the strengthening of AIDS prevention and care activities at all levels with particular attention to the special needs of women.

The risk associated with disease that causes genital ulcers, such as syphilis, chancroid and herpes HIV transmission increases 10 fold and disease causing discharges especially gonorrhoea, chlamydial infection and trichomoniasis is up to 4 fold, the community at large is unaware of these facts and also that HIV is not treatable unlike STDS.

As a result HIV infection rate among people seeking treatment for STDS has increased from 9% in 1991 to 17% in 1992 in the city of Pune while the rate in Calcutta and Bombay were 5% and 25% respectively for HIV among STD patients in 1993.

In India so far nearly 25 lacs samples have been screened which has yielded 1829 (1995 May) seropositive individuals giving a rate of 7.29/thousands.

The AIDS cases detected so far 1139 of whom 263 are females. But these figures represents only the tip of the iceberg due to under diagnosis and low reporting. WHO estimate that India has the largest number of infected individual among SE Asian countries.

*Global trends: January 1 1992 WHO estimated that these were about 13 million human immuno deficiency virus infected person in the world with almost two thirds residing in Sub Saharan Africa. More than one million HIV infected persons live in North America, one million in South America
and more than 7,00,000 in Western Europe. Where as more than 90% of the African infection are estimated to have been acquired heterosexually the most recent data regarding the prevalence of this mode of transmission indicate rates of 9% in North America, 14% in Western Europe and 24% in Latin America.

**AIDS Surveillance:**

Govt. Of India has established a network of surveillance centres in the country to screen high risk group (Govt. Of India 1991). This includes establishment of nine referral centres (e.g. National institute of virology Pune, Christian Medical College Vellore, All India Institute of Medical Sciences, New Delhi and National institute of Communicable Diseases Delhi) Where higher level diagnostic facilities are available. By the end of 1992 Govt. Of India has established 62 surveillance centres for screening persons practicing high risk behavior. Realising the gravity of epidemiological study of HIV in the country a separate wing “National AIDS control organisation” has been set up under the Ministry of Health and family welfare.

The WHO had launched a global programme on AIDS on Feb. 1 1987 to provide global leadership and to suppose the development of National AIDS programmes. Centre for disease control (1982) found that the first AIDS (Acquired immune deficiency syndrome) in a women was reported in U.S.A. in 1981 and perinatal transmission was described in 1982. In 1983 first case of heterosexual transmission was reported in the centre.
Jovaicas et al (1985) gave termination of pregnancy in an HIV antibody positive women at 20 wks gestation and foetal sample were found to contain HIV. So HIV seropositive among pregnant women is therefore a great risk to unborn child.

Menez Baulisha et al (1986) the following 3 cases reported clear evidence of intrauterine transmission. Sprecher et al (1986) detected HIV antigen amniotic in fluid and foetal tissue from a pregnancy terminated at 15 weeks of gestation in a women with stage “AIDS” and Kaposi’s sarcoma.

Vigneron et al (1987) demonstrated 640 pregnant women screened for HIV seropositivity 25 (3.8%) were seropositive for HIV antibody and five of the seropositive pregnant women had clinical or biological evidence of AIDS related complex.

Minkoff et al (1987) studied that HIV -1 infection itself may adversely affect pregnancy remains controversial while some studied from North America and Europe demonstrated an adverse effect of HIV-1 infection on pregnancy out come other failed to confirm these finding because most of these studies were uncontrolled. Their relevance to other population of pregnant HIV infected women remains unclear.

Gloeb et al (1988) report of 50 HIV seropositive pregnant women found that 35 seropositive women have complicated perinatal courses and most commonly premature labour or infections complication. premature labour
complicated 35% of pregnancies among HIV infected women.

Heath et al (1988) an analysis of 2200 stored serum sample collected at routine antenatal clinics over a period of nine months was carried out six positive results were obtained a seroprevalance rate of 0.3% Krarivski et al (1988) Barbucci et al (1990) when analysed at a time for one year only one seropositive individual had been identified. Glach and Coellegeues (1988) followed the clinical course of 50 HIV infected women antepartum and or post partum. Three patients died of complications related to AIDS. Two of these were asymptomatic when first seen in the course of their pregnancy and two developed pneumocystis carinii pneumonia and died during pregnancy and third developed Toxoplasma gondii encephalitis at 18 weeks gestation and died four months after delivery. Another two patients developed AIDS related symptoms in the third trimester. An additional four developed candidiasis out of which three had CD4 count under 160 nm 3 at delivery.

Jean pape (1988) obtained information about sexual activity on 151 HIV seropositive and 131 seronegative spouse of male AIDS followed from 1983 to 1986. As a result 14% (22/151) of the seropositive sex partner and 12% (15/131) of the seronegative sex partners have become pregnant during a mean period of follow up of 15 months (range 2-36 months) and 18 months (range 2-38 months) respectively.

27% of the seropositive pregnant women (6/22) had a miscarriage compared to 13% (2/15) of the seronegative pregnant women.
In addition to (9/97) female AIDS (11/%) followed for 5 months (range 1-24 months) become pregnant.

Coles et al (1988) studied in France 2/4674 pregnant women revealed that as many as 2 births or abortions per day involves women having HIV antibodies.

According to available information the procedure of France gynaecologist obstetrician is as the following:-

1. During the first trimester of pregnancy, we recommend an abortion after having informed the women of the consequences of the presence of the virus.

2. During the second trimester of pregnancy, a free choice is left open for the women to decide whether she desires an interruption of pregnancy or to carryout her pregnancy. In few case foetal monitoring is essential.

3. During the third trimester foetal state is monitored, delivery is carried out. Cesarean are only done where there are obstetrical indication. 50% women have had abortion. 50% have delivered.

Embres (1989) studied HIV infection on foetus growth failure may be seen in 75% or more of infected infants. It has been suggested that deformities including microcephaly, ocular hypertension, prominent forehead, flat nasal bridge, long palpebral fissures, blue sclera and patulaus lips may be associated with congenital infection, however the relationship of these features to HIV infection has been challenged.
Ryder et al (1989) studies from African however describe an unfavourable effect of HIV-1 infection on pregnancy outcome including foetal wastage prematurity, low birth weight, still birth and neonatal death but not upon embryopathy on congenital abnormalities.

Bigger et al (1989) have demonstrated that HIV antibody positive women exhibit a greater and more sustained fall in CD4 lymphocytes cell counts during compared with seronegative pregnant women.

Delfraissy et al (1989) believe that pregnancy may accelerate the progress of HIV infection. But Bradbeen 1989 does not believe it.

Lakshmi & Gururaj kumar (1989) studied 2 pregnant women one of 400 were found to be seropositive for HIV antibody by ELISA, the positive rate being 0.5% both of them were primigravida in mid term pregnancy come from urban area and belong to low socioeconomic status. There was also history of multiple sexual partners drug abuse, and blood transfusion in the past and their husband had history of STDs.

Braddick et al (1990) suggested an association an association between natural HIV infection and adverse pregnancy outcome including still birth, low birth wt. And neonatal mortality. (lepage et al 1992) said that HIV infected infants of lower birth weight than non infected infants born to infected mother. a finding that was not statistically significant.
Minkoff et al (1990) said that the HIV infection itself does not seem to have a major direct effect on pregnancy outcome.

Kell et al (1991) reported a 26 year old Ugandan woman presented 10 weeks gestation in her second pregnancy to a district general hospital casualty department complaining of shortness of breath or exertion, fever and productive cough for 1 week, six years back she had an uneventful pregnancy, as she had arrived in Britain as a political refugee. Only two weeks before initial physical examination revealed Tachypnoea (70/nin) and Pyrexia her chest x-ray was suggested of an interstitial Pneumonia. Arterial blood gases on air showed a po2 of 10.0kpa. She was treated with intravenous ampicilline and erythromycin for 3 days but she continued to deteriorate. A blood sample was then taken to test for HIV antibody after the patients was counselled the result was found to be positive. This case represents the first maternal death associated with HIV infection to be reported in Britain. Diagnosis of HIV seropositivity affect the outcome.

European collaboration study group (1992) investigated 721 children born to 701 HIV infected mother and found a 14.4% vertical transmission (Mother to children) and transmission was associated with CD4 count <700, there was associated relationship between transmission and preterm delivery (<34 weeks gestation).

Johnstone et al (1992) believes that the pregnancy is associated with mild impairment of cell mediated immunity and increased virulence of some infections.
Johnstone et al (1992) demonstrated 5 pregnant women and showed that survival time was not obviously reduced by the conjunction of pregnancy with AIDS. Kenyan study shows that the outcome of AIDS in pregnancy is poor. In this study maternal mortality with AIDS was high with 60% of birth occurring in perinatal period.

European collaboration study group (1992) demonstrated in 30 Nov. 1992 1200 mother child pair were enrolled (19 European centres). Children with known HIV infection (born to women with HIV positive antibody at or before the delivery) status were available for present analysis. All multiple pregnancies and children whose HIV infection status was still indeterminate were excluded.

3 Mothers were known to receive Zidovudine during pregnancy and all 3 infants were uninfected.

Mask w. Kline et al (1993) said 142 infants have been referred to our centre because they were born to HIV infected mothers. 24 (17%) of these infants have been lost in follow up and 3 (2%) have transferred to other institutions 30 (21%) infants are younger than 15 months of age and have indeterminate HIV infection status of the 85 infants whose infectious status is known, 7 (20%) have confirmed HIV infection and 68 have seroconverted to HIV and lack of evidence of infection. 14 HIV infected infants currently are symptomatic or have died and 3 are asymptomatic.

Thorgecharoen et al (1993) studied from January 1991 to Dec. 1993, 24856 parturients registered for prenatal care at Ramathibodi Hospital and all were tested for the first for anti HIV screening 91 (0.36%) were seropositive by ELISA
and western blot confirmation test at the first screening. 8 women were found to be seropositive at 28-32 weeks gestation with the rate of 0.03 per 100 seronegative test at the first screening. One woman was seropositive by ELISA and western blot at the screening.

132 seronegative pregnant women were recruited in the control group.

The flow chart diagram of seropositive and seronegative group. At first anti HIV screening these were 91 seropositive and 182 seronegative pregnant women. After post term counselling 78 seropositive cases (85.7%) decided to terminate the pregnancy (51% in first trimester and 49% in second trimester). 13 women continued pregnancy for those who had the pregnancy terminated there was 2.6% with post abortal endometriestis and there was no serious complication. At the second anti HIV screening their were 8 additional seropositive cases giving a total of 21 deliveries. There were 11 cases of spontaneous abortion (6.1% in seronegative group).
Seropositive

First
Spontaneous
Screening
Abortion (N=11)

Continue Pregnancy

N = 13

Seronegative

(N=182)

Continue Pregnancy

N = 171

Second Seroconversion screening
N = 8

Continue Pregnancy

Temmernan M. et al (1991) suggested the outcome of AIDS in pregnancy is poor. Pregnancy could increase the probability of AIDS defining illness becoming fatal because of delay in diagnosis. Another contributing factor is that the HIV infected women were from very low socioeconomic status receiving inadequate and inappropriate Medical care. HIV infected women had significantly higher rate of miscarriage and preterm delivery (P < 0.001). The mean birth weight < 2.5 kg was significantly more of these women delivered at a gestation age of less than 37 weeks.

Prematurity was more common in mother with AIDS or AIDS related sign and symptoms than in the asymptomatic HIV infected group and in seronegative control, these were no congenital abnormalities or still birth in either group.
however these were 7 cases of intrauterine foetal death due to AIDS related maternal deaths (HIV positive (N=160) negative N=164).

Caroline et al (1994) said that at least two hypothesis can be suggested for the effect of pregnancy on HIV diseases progression, first the short term risk of developing AIDS or HIV related disease may be much higher during pregnancy, with no residual excess risk once the pregnancy is over. The immediate risk of developing AIDS returns to what it would have been if the pregnancy has not occurred, Alternately pregnancy might accelerate HIV disease progression irreversibly leaving women who have been some time if the pregnancy have never occurred multiple pregnancies would produce a cumulative detrimental effect on risk.

Tsemmerman et al (1994) studies from africa suggest an association between low birth weight and or asymetrically and maternal HIV infection. In 1988 in Narobi showed an independent association between HIV-1 seropositivity and being born small for gestational age and premature.

European collaboration study group (1994) suggest the occurrence of vertical transmission of HIV-1 during the antepostum, intrainству and post partum periods vinal antigenic acurrity has been demonstrated in foetal amniotic cells as early as 15 weeks of gestation.

Mauri et al (1995) reported that the gestational age was significantly reduced in infected and uninfected opiate addicts (P<0.0001) where as maternal weight gain was lower in both HIV carrier group as well as in seronegative opiate users (P<0.001). Incidence of miscarriage was higher in both
infected groups, although statistical significance was reached only in simple HIV seropositive (P<0.05). The slightly higher percentage of threatened preterm labour and caesarean section due to foetal indications found in simple HIV carrier further increased in both opiate addicted group, who showed a significant rise in the incidence of preterm labour (P<0.05) the percentage of infected children was 33.3% and 31.6% in simple HIV seropositive and HIV seropositive opiate user respectively.

Herman et al. (1995) reported a 34 - years old American women at 37 weeks estimated gestational age by early sonography, was seen in our obstetric triage area complaining of malaise and rupture of membranes. The patients admitted to using crack cocaine for the last 5 days and a crack house, she developed malaise and fever three days earlier and progressively worsening abdominal pain about six hour prior to admission.

The patient was known to HIV positive as confirmed by western blot method and had been followed in the infectious disease clinic and absolute CD4+ lymphocytes count and 1 week prior to admission was 36 mm³.

John et al. (1995) reported 1 seropositive women and 3 seronegative women reported refusal of health care after the HIV test reason was citing and discrimination.

Correlation between TORCH and HIV infection.
Correlation between HIV and Toxoplasma.

Women infected with HIV are at risk for transmission of Gondii infection to the foetus both of they are
seronegative for T.gondii antibodies and acquire T.gondii infection during pregnancy and if they are seropositive for T.gondii antibodies and suffer reactivation of their latent T.gondii infection because of immune deficiency from HIV infection.

Mitchell et al (1990) revealed a congenital transmission rate for women who are dually infected with HIV and T.gondii that was remarkably higher when compared to non HIV infected, Toxoplasma seropositive pregnant women.

All infants with congenital Toxoplasma born to mother who were HIV infected also were infected with HIV.

The initial clinical presentation of congenital Toxoplasma in HIV infected infants is similar to that and non HIV infected infants but appear to run a more rapid and progressive course. The infants often appear normal at birth in the ensuring month they fail to gain weight or develop appropriately. The majority develop multisystem organ involvement including CNS, Heart, Lungs.

Correlation between Toxoplasma and Rubella.

Chitra Raghunandan (1993) said the present study was conducted in 25 cases of missed abortion between 6-20 weeks of gestation and 25 cases of unexplained intrauterine death between 21-40 weeks of gestation, 25 cases of MTP and 25 cases of normal pregnancy were taken as control. Their sera were tested by ELISA for IgM specific antibodies to Toxoplasma gondii and rubella.
The 50 cases of study group, 16 cases (32%) showed antibodies to Toxoplasma, (6 cases) Rubella, (1 case) and Syphilis (9 cases) as compared to all the control cases who were seronegative (P<0.01). There were no cases with more than one etiological agent. All 6 cases seropositive for Toxoplasma as well as 1 case of rubella were associated with missed abortion were 8 cases out of 9 cases of syphilis associated with late foetal death.

**Corelation between Toxoplasma gondii, Rubella cytomegalovirus and Herpes simplex virus.**

P. Prabhaker (1990) seroprevalence of Toxoplasma gondii Rubella virus, cytomegalovirus Herpes simplex virus infection and syphilis were determined in order to assess and the immunosusceptibility foetus in American Pregnant women in 1986 the positive rate were 57% (T. gondii) 69% (rubella) 97% (CMV) 91% (HIV) and 4.9% (syphilis) respectively. The rate of reactivity for syphilis ranged from 2.1% in Kingston and St Andrew at 7.3% in rural Parishes. The seropositivity for syphilis ranged from 21% in the seropositive rate for Rubella was over 50% parishes, the highest being 85% in St Thomas.

The seroprevalence of T. Gondii was lowest in Trelawny (37.5%) there were no significant difference in seropositivity of CMV and HSV infected between women various parishes.