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Human Immunodeficiency Virus Transmission

Goselle Obed Nanjul^{1,2}

¹*School of Biological Sciences, Bangor University*

²*Applied Entomology and Parasitology Unit, Department of Zoology, University of Jos,*

¹UK

²Nigeria

1. Introduction

Human Immunodeficiency Virus (HIV) is the causative organism of AIDS which has become one of the greatest public health challenges faced by mankind. AIDS was first identified in 1981 in Los Angeles, USA. Two types of HIV exist presently- HIV-1 and HIV-2 (Alizon et al., 2010; Adoga et al., 2010). HIV-1 was first isolated in the early 1980s (Barre-Sinoussi et al., 1983) and linked as causative agent of AIDS (Gallo et al., 1984). HIV-2 which is similar to HIV-1 was later identified in the developing world (Clavel, 1987, Clavel et al., 1986), but found to be less virulent and can differ in its response to antiretroviral agents. HIV-1 is classified into three groups [M, N and O] based on the genetic diversity. Group M (major) has 10 subtypes (A-J), and Group O (outlier) represents a number of highly divergent strains (Carr et al., 1998; Jassens et al., 1997; Chen et al., 2010). Francois Simon and his group reported a group N of HIV-1. Despite the phenotypic classification of HIV-1 into subtypes, the number of sequenced isolates remains limited (Sharp et al., 1994). Both strains are spread in the same way and have the same AIDS causing consequences. While HIV-1 has been reported to have a shorter incubation period of 7-10 years, HIV-2 is considerably longer and often less severe (Barre-Sinoussi, 1996; WHO, 1989).

HIV infection is usually followed by a chronic progressive destruction of the immune and neurologic system (Price, 1996), which if not managed leads to the possible invasion and establishment of multiple opportunistic infections (Lindo et al., 1998; Pozio et al., 1997) and malignancy (Schulz et al., 1996). Although on average, an infected individual spends several years without manifesting the disease, AIDS has always been certain. The time from infection to AIDS varies widely between individuals, from a few months to as many as 20 years with existing evidences accepting that 50% of individuals progress to AIDS in 7-10 years and this has been accepted as the incubation period of the virus (Del Amo et al., 1998; WHO, 1994).

2. Portals of HIV transmission

The concentration of virus in a body fluid and the extent of exposure to body fluids determine to a great extent the transmission of a virus. Jaffe and McMahon-Pratt (1983) first indicated in their Epidemiological studies conducted in 1981 and 1982 that the major channel of transmission of AIDS were intimate sexual contact and contaminated blood. Gottlieb et al (1981); Masur et al (1981); Siegal et al (1981); Callazos et al (2010); van

Griensven and de Lin van Wijngaarden (2010) all described the syndrome in homosexual and bisexual men and, intravenous drug users, while Harris et al (1983); Padian et al (1991); Cameron et al (1989); Quinn et al (2000) and Decker et al (2010) recognised their mode of transmission through heterosexual activity. Evidences later showed that transmission recipients and haemophiliacs could contract the illness from blood or blood products (CDC, 1982; Peterson, 1992; CDC, 2010) and newborn infants get infected from their mothers' (Ammann et al., 1983; Scarlatti, 1996; Brookmeyer, 1991; Landesman, et al., 1996; Goedert et al., 1989; Mackelprang et al., 2010). Brookmeyer (1991); Stoneburner et al (1990) all agreed that the three principal means of transmission – blood, sexual contact and mother-to-child have not changed which could be attributed to a greater degree to the relative amount of the virus in various body fluids.

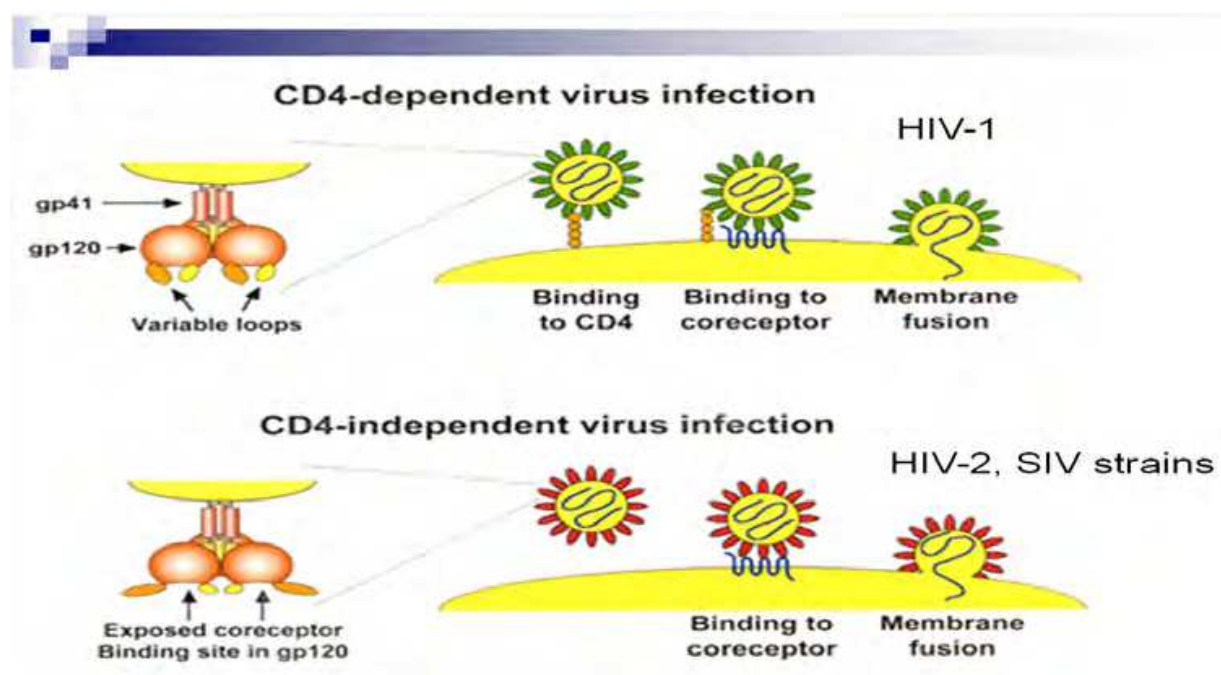


Fig. 1. Diagrammatic representation of HIV-1 and HIV-2 showing their dependent and independence on CD4+ [Courtesy-]

HIV is present in semen (including pre-seminal fluid), vaginal/cervical secretions and blood, breast milk expressed through feeding; organ donations; sharing infected objects (needles, tattoos and piercing) which are the main vehicles through which the virus is transmitted (Kim et al., 2010; Yu et al., 2010; Suligoi et al., 2010; Pruss et al., 2010 and Baggageley et al., 2010). The virus may also be present in saliva, tears, urine, cerebrospinal fluid and infected discharges, but these are not vehicles of which HIV is spread. Epidemiological survey do not support transmission through water or food, sharing eating utensils, coughing or sneezing, vomiting, toilets, swimming pools, insect bites, shaking of hands or other casual contacts, hence there is no public health reason for discrimination and or restrictions.

A study of French hospital patients by Grabar et al (2009) found that approximately 0.5% of HIV-1 infected individuals retain high levels of CD4+ T-cells and a low or clinically undetectable viral load without anti-retroviral treatment. These individuals are classified as HIV controllers or long-term non-progressors.

For conveniences, we will share the mode of infections into: Sexual and Non-sexual.

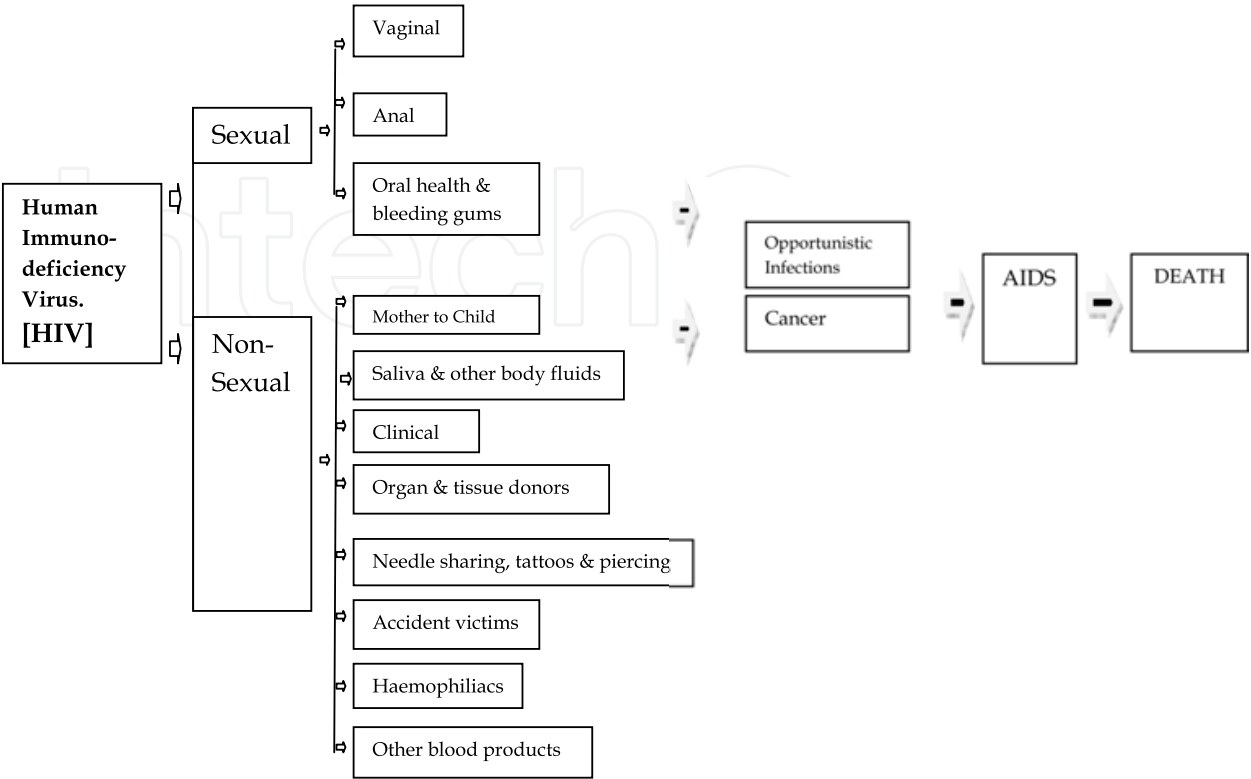
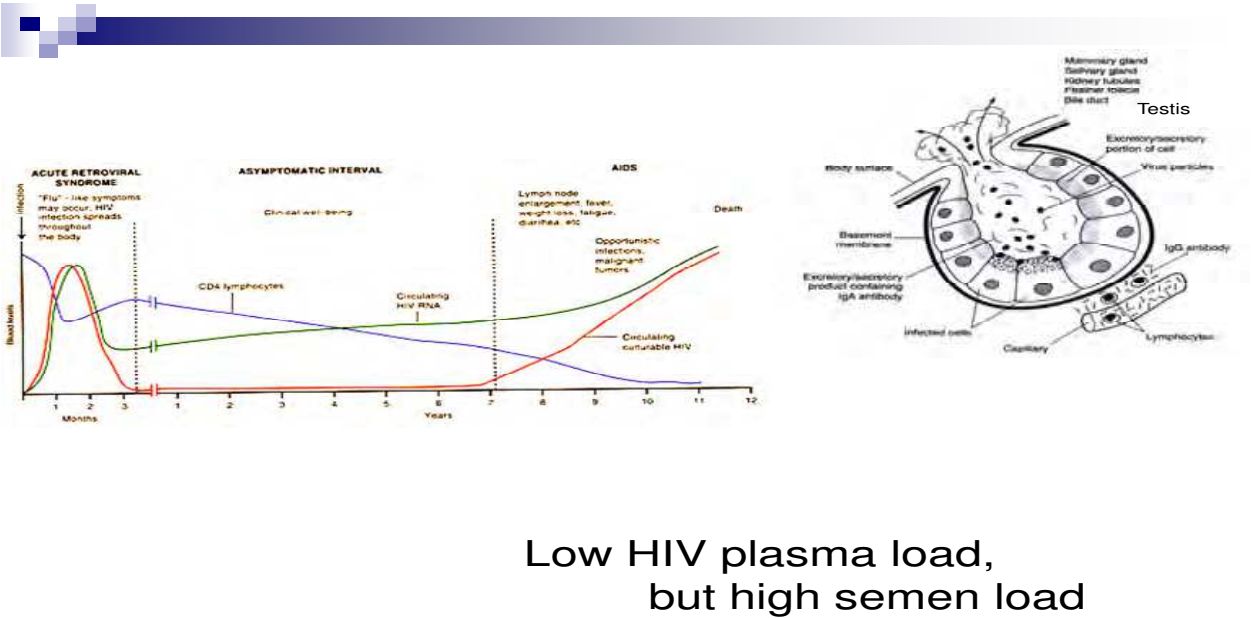


Fig. 2. Routes of Transmission of Human Immuno-deficiency Virus. [HIV]



Low HIV plasma load,
but high semen load

Fig. 3. Levels of HIV load in semen [Courtesy:...]

3. Vertical or Mother To Child Transmission (MTCT)

The major source of paediatric infection of Human immunodeficiency virus one (HIV-1) is from mother to child. Since the first reported case of HIV-1 transmission in children in 1983, the global pandemic has had a serious impact on the health and survival of children. Transmission rates have been reported to be about 14% in industrialised countries and about 35-45% in developing countries especially in Africa (Bryson, 1996; Reinhardt et al., 1995).

It was estimated that MTCT accounts for over 1.5million HIV infection in children (Burton, 1996) with the WHO projecting between 5-10million child infections through MTCT during the next decade. HIV-2 though is related to HIV-1 is less readily transmitted from mother to child, this could be attributed to their differences which influences pathogenecity, natural history and therapy so that their susceptibility to antiretroviral therapy (ART) follows different mutation pathways to develop drug resistance (Mamata and Merchant, 2010).

According to Wollinsky et al (1992) as quoted by Pasquier et al (1998), the transmission of HIV-1 from mother to child occur *utero*, *intrapartum*, or postnatally by breastfeeding and a fourth dimension as reported by Pasquier et al (1998) which involves the transmission of multiple maternal variants to the infant and a rapid, fatal outcome in the child and the development of an HIV-based clinical disease in children seems to be correlated with the timing of the vertical transmission.

Infection in about two-thirds of children are thought to have occurred at the terminal end of pregnancy or at delivery with the disease progressing slowly; while in one-thirds, it is thought to progress rapidly to AIDS with increased indices of viral replication (De Rossi et al., 1998), these children appear to have been infected during pregnancy.

Infected children with slow progression to AIDS have a higher viral diversity than children who progress rapidly as evidenced in molecular variability studies (Halapi et al., 1996; Strunnikora et al., 1995) as reported in Adults (Delwart et al., 1997; Pasquier et al., 1998).

Although progress has been made in recent years in the curbing of MTCT, the mechanisms and timing of transmission remains uncertain and the relative contributions of each of the three modes of transmission is still not well defined. Bryson et al (1992) proposed that in most non-breastfeeding population; the lack of detection of virus in the child at birth might indicate that contamination took place at or shortly before delivery while detection of virus at birth indicates *utero* contamination. Evidences for both early and late *utero* transmission have been documented (Peckham and Gibb, 1995; Kuhn and Stein, 1995). Most prior estimates and hypothesis seem to agree that transmission usually occur during the *intrapartum* HIV exposure just as premature infants.

Perinatal or *Antepartum* HIV transmission has been documented as a route of infection estimated to occur in 13-30% of infants delivered to HIV-1 infected mothers (Andiman et al., 1990).

High proviral DNA/ or RNA concentration of virus is a risk factor for the transmission of HIV-1 from an untreated mother to infant. The reduction in such transmission after zidovudine is only partly explained by the reduction in plasma levels of viral RNA. To prevent HIV-1 transmission initiating maternal treatment with zidovudine is recommended regardless of the plasma level of HIV-1 RNA or the CD4+ Count (Sperling et al., 1996). Because of the different mutation pathways to develop drug resistance, pregnant women with detectable HIV-2 should be ideally managed using a Highly Active antiretroviral therapy (HAART) regimen to which the virus is sensitive. Non-nucleoside Reverse

Transcriptase Inhibitor (NNRTIs) and Fusion Inhibitor Enfuvirtide have no activity against HIV-2 and in the light of the current albeit limited data, zidovudine mono-therapy should not be used. These factors make it crucial that proper selection of and adherence to the first antiretroviral combination regimen is in place in order to achieve a successful treatment response. Though of recent, a combination of Combivir and nevirapine is given to mothers to prevent transmission of HIV to children. The Emergency Lower Segment Caesarian Section (ELSCS) could be planned at 38 weeks of gestation with regards to the mode of delivery if the viral load is undetectable or the mother is either symptomatic or has low CD4 cell count. HIV is present in breast milk and postnatal transmission via breastfeeding is an important component of MTCT in Sub-Saharan Africa (Kreiss, 1997). World-wide, an estimated one in three of vertical transmission may be due to breastfeeding with above 12 months of age carrying higher risk (Bulterys et al., 1995). Kuhn and Stein (1997) demonstrated that under certain conditions prevailing in specific settings in developing countries, breast feeding for six months would be preferable to breast feeding beyond this age. Breastfeeding has been reported to account for 5-15% of infants becoming infected with HIV-1 after delivery (ECS 1991; Ryder et al., 1989; Mok et al., 1989). Although the placental entry of some infections is a critical aspect of these infections, the role of placental cells and the mechanism by which pathogens pass from the maternal to the foetal circulation varies. The placenta provides a barrier that prevents transmission of some viruses, but allows others to reach the foetal circulation. Mother to foetus placental transmission of some viruses occurs through transcytosis across placental cells. The placenta may also act as a reservoir in which virus replicates before reaching the foetus. Placental transmission of HIV-1 is a complex incompletely understood process which requires advanced studies (Al-husaini, 2009). The antiretroviral therapy, zidovudine (ZDV) is metabolized into its active form in the placenta (Qian et al., 1994). ZDV inhibits HIV replication within placental cells. To reach the foetal circulation, HIV-1 should cross the trophoblastic placental barrier (cytotrophoblasts and syncytiotrophoblasts). Blood borne maternal pathogens that arrive at the uteroplacental circulation and intervillous space may reach the foetus through the villous capillaries. HIV-1 has been detected on both the maternal and the foetal parts of the placenta. HIV-1 experiences replication in the placenta. The virus may cross the trophoblastic barrier by endocytosis, or by an injured villous surface. However, superficial breaks in syncytiotrophoblast cells do not radically affect the vertical transmission of viruses (Burton et al., 1996). The reverse transcriptase enzyme of HIV-1 is important in the life cycle of the virus by converting the single-stranded RNA genome into double-stranded DNA that integrates into the host chromosome. There is a lower degree of viral heterogeneity in transmitting mothers compared with nontransmitting mothers (Sundaravaradan et al., 2005).

Human chorionic gonadotropin (hCG) has been shown in vitro to inhibit reverse transcriptase and to block viral transmission between virus-carrying lymphocytes and placental trophoblasts (Bourinbaier and Lee-Huang, 1995). However, role of hCG in protecting the foetus from vertical transmission HIV-1 needs to be studied. In summary, the restricted heterogeneity of HIV-1 in the infected mothers is more likely associated with lack of vertical transmission (Al-husaini, 2009).

As access to services for preventing the mother-to-child transmission of HIV has increased, the total number of children being born with HIV has also decreased. An estimated 370 000 [230 000–510 000] children were newly infected with HIV in 2009 (a drop of 24% from five years earlier)[UNAIDS, 2010].

4. Risk factors for vertical transmission of HIV

Documented evidence primarily based on PCR and virus culture studies or co-culture studies but short of serology which revealed maternal antibodies present in infants at birth showed that transmission of HIV from mother to child appears to occur in 11-60% of children delivered by HIV-positive mothers but reasons for the wide variations in virus transmission and sources of virus in newborn which could have provided approach to prevention are not known (Ades et. al., 1991; Courgnaud et. al., 1991; Lindgren et. al., 1991; Newell et. al., 1992; Scarlatti et. al., 1991; Tovo and Martino, 1988; Oxtoby, 1990; Rogers et. al., 1991).

Maternal, viral, obstetric, foetal, infant factors all affect transmission making it essentially multifactorial. Frequency of sexual activity, 'hard' drug ingestion during pregnancy, unprotected sexual intercourse, cigarette smoking during pregnancy, lack of adherence to drugs, HIV disease, degraded maternal immunocompetence or prolonged rupture of the amniotic membranes before delivery (Havens et al., 1997; Turner et al., 1996; Bryson, 1996; John and Kreiss, 1996; Lambert, 1996; Glenn and Dietrich, 1993).

The maternal factors involve transmission through the placenta to the unborn child, at the time of labour and delivery, or through breast-feeding. (CDC HIV/AIDS surveillance, October, 1989), seroconversion during pregnancy, advanced stage of the disease with high viral load and low immunity, concomitant malnutrition, micronutrient deficiencies, sexually transmitted diseases, no or suboptimal therapy; in the intranatal period, risk factors for increased transmission are mode of delivery, prolonged contact with maternal blood or cervicovaginal secretions, prolonged rupture of membranes, chorioamnionitis, invasive procedures like episiotomy, foetal scalp electrode, instrumental delivery; thin skin, susceptible mucous membranes, immature immune functions and low levels of maternal antibodies make prematurity a risk factor for increased transmission. In the postnatal period, risk factors are breast feeding, feeding with cracked nipples/mastitis, mixed feeding, new seroconversion of the mother, high viral load, low CD4 cell count; In the absence of any intervention, rates of MTCT of HIV-1 can vary from 15 to 30% in developed countries and increase to 30 to 45% in developing countries, the difference mainly attributable to infant feeding practices that comprise almost universally of breastfeeds for prolonged duration (De Cock et al., 2000 as quoted by Mamata and Merchant, 2001).

The foetus and mother circulatory systems though different, there still exists tiny mixing of blood that could serve as portal for the flow of infected maternal white blood cells or the AIDS virus in the maternal serum to be transmitted to the foetus with a confirmation found in the foetal tissues affirming such spread (CDC HIV/AIDS surveillance, October 1989; Glenn and Dietrich, 1993).

Bruising, abrasions and local swelling could occur to the baby and mother during labour owing to a great deal of trauma which produces visible and microscopic openings that could allow the virus to penetrate blood stream of infant. Another means of infection could be experienced or seen when the mother's perineum tears or if she receives an episiotomy which might lead to a large amounts of blood ingested by the baby or might get into the baby's mouth, eyes, rectum or vagina.

Glenn et al (1993) reported that breastfeeding is another means of risks exposure and it has been confirmed in the spread of hepatitis B from mother to infant and hepatitis B and AIDS

as well which are thought to occur when the infant ingests the mothers blood through a cracked and bleeding nipples.

Other known correlates include high maternal plasma viremia, advanced clinical HIV disease, degraded maternal immunocompetence or prolonged rupture of the amniotic membranes before delivery. Others include vaginal delivery process and prematurity of low birth weight of the neonate (Bryson, 1996; John and Kreiss, 1996; Lambert, 1996).

High frequency of sexual activity and “hard” drug injection during pregnancy had previously been identified, along with unprotected sexual intercourse during pregnancy as certain behavioural risk factors for mother-to-child-transmission (Bulterys et al., 1997; Bulterys and Goedert, 1996). Firstly, unprotected intercourse might increase the concentration of strain diversity of HIV-1, particularly in the birth canal where ejaculated virus could be partially sequestered. Secondly, frequent intercourse might increase inflammation of the cervix or vagina either micro abrasion or if unprotected, by STDs. Third, frequent intercourse might increase the risk of chorioamnionitis or otherwise alter the integrity of the placenta (Bulterys and Goedert, 1996). Matheson et al (1997) found that continued drug users had significantly higher mother-to-child-transmission rates in maternal drug use during pregnancy. However, this was confounded by other variables such as premature delivery, prolonged membrane rupture, zidovudine non-use and unprotected sexual intercourse.

In the USA, cigarettes’ smoking during pregnancy has been identified as independent risk factor for mother-to-child-transmission. The effect was greatest among women with critical evidence of more advanced HIV disease (Turner et al., 1996). Intensive nurse care management in supporting zidovudine use in women with HIV infection and their infants is a proven effective method in decreasing mother-to-child-transmission (Havens et al., 1997).

MTCT of HIV is influenced by multiple factors. Known correlates include high maternal plasma viremia, advanced clinical HIV disease, degraded maternal immunocompetence or prolonged rupture of the amniotic membranes before delivery. Others include vaginal delivery process and prematurity of low birth weight of the neonate (Bryson 1996; John and Kreiss, 1996; Lambert, 1996).

Results from zidovudine therapy to bridge MTCT have improved understanding of the pathophysiology of MTCT. First, the reduction in plasma viremia and MTCT (from 25.5% to 8.3%) by treating the mother and neonates suggests that relatively small changes in maternal viral load might have substantial effects on MTCT (Bulterys and Godert 1996; CDC, 1994). Secondly, cleaning of birth canal with chlorhexidine had no overall effect yet apparently did reduce MTCT for one subgroups of high-risk deliveries; those after 4hrs of membrane rupture (Scarlatti, 1996).

Maternal immunologic and virologic factors such as quantitative HIV-1 RNA (though insufficient) are strongly correlated with Mother-to-child-transmission. When stratified by the stage of HIV disease, the only group with significant association between viral load and mother-to-child-transmission were AIDS-free women with high CD4⁺ Counts. The interactions of virus burden and maternal immune status has also demonstrated that CD4⁺, CD8⁺ cell subsets are percentages of CD8⁺ cell subsets (e.g. activation markers CD8/CD38 and CD8/DR) were all associated with vertical transmission. Women in the highest CD4⁺ cell percentage quartile or the lowest CD8⁺ cell percentage quartile had only less than or equal to 4 percent of mother-to-child-transmission (Njoku, 2004).

5. Parental, saliva and other body fluids

Prior to Groopman and Greenspan (1996) report of oral manifestation of AIDS which increases the potentials of HIV transmission through several lesions which form exists for virus into the saliva, it was assumed that about 10% of both free virus and infected cells report in saliva were not very important in the spread of HIV (Groopman et al., 1984).

Dean et al (1988) and Mundy et al (1987) reported none or low level of pathogens in urine, sweat, breast milk, bronchoalveolar lavage fluid, amniotic fluid, synovial fluid, faeces and tears which were not thought to be important source in virus transmission (Fujikawa et al., 1985), but this assumption has also changed with the report of Groopman and Greenspan (1996); Amory et al. (1992); Scarlatti (1996); van da Perre et al. (1991). Though not a natural source of HIV transmission, cerebrospinal fluid (CSF) in neurologic patients have been shown to contain large amount of virus when compared to other body fluids (Hollander and Levy, 1987; Ho et al., 1989).

6. Organs, blood, tissue donors and occupational health workers

Prior to 1985 (PPHS/MMWR, 1985; MMWR, 1985), when screening of blood, organ and tissue donors for HIV-1 antibody became available, several reports have documented the transmission of HIV-1 by transplantation of kidney (MMWR, 1987; Kumar et al., 1987; Erice et al., 1991; Schwartz et al., 1987; Prompt et al., 1985; L'age-Stehr et al., 1985; Neumayer et al., 1987; Quarto et al., 1989; Carbone et al., 1988), liver (MMWR, 1987; Kumar et al., 1987; Erice et al., 1991; Schwartz et al., 1987; Prompt et al., 1985; L'age-Stehr et al., 1985; Neumayer et al., 1987; Quarto et al., 1989; Carbone et al., 1988; Samuel et al., 1988), heart (Erice et al., 1991; Dummer et al., 1989), pancreas (Erice et al., 1991), bone (MMWR, 1988a) and possibly skin (Clarke, 1987) and In most cases involving donors whose serum had not been tested for HIV-1 antibody (MMWR, 1987; Kumar et al., 1987; Erice et al., 1991; Schwartz et al., 1987; Prompt et al., 1985; L'age-Stehr et al., 1985; Neumayer et al., 1987; Quarto et al., 1989; Carbone et al., 1988; Samuel et al., 1988; Dummer et al., 1989; MMWR, 1988a; Clarke, 1987).

As proposed by Simonds et al (1992), approaches to prevention could include: the screening of prospective donors and laboratory markers for HIV1 infection (MMWR, 1985); the inactivation of HIV-1 in allograft through processing techniques (Hilfenhaus et al., 1990; Kitchen et al., 1989; Wells et al., 1986) and the quarantining of tissues from living donors until repeated antibody testing more definitely excludes the possibility of subsequent seroconversion in the donor (MMWR, 1988a; MMWR, 1988b).

The U.S. Centers for Disease Control and Prevention (2002) reported that in the health care industry there have been 57 confirmed cases and an additional 139 possible cases of health care workers in the U.S. who have become HIV positive from exposure to HIV in the work place. The Canadian HIV/AIDS Legal Network (2001) has also reported two of such cases in the laboratory workers and one health-care provider in Canada.

7. Horizontal (heterosexual) transmission

These could be through unprotected and protected sexual process. Ma et al (2010) reported that the probability of unprotected heterosexual transmission may vary with population and be influenced by many factors, these could include: the type of sex (Mastro et al., 1994; De Vincenzi, 1994; Varghese et al., 2002); bleeding during intercourse (Royce et al., 1997),

semen viral load (Gupta et al., 1997; Tachet et al., 1999; Kalichman et al., 2008; Butler et al., 2008), stage of HIV infection (Mastro et al., 1994; Fauci et al., 1996; Wawer et al., 2005), co-morbid sexually transmitted diseases (Royce et al., 1997), vaginal or anal canal, co-occurring psychosocial risk factors (Safren et al., 2010).

Sexual forms of transmission are seen as a major portal of entry of HIV as 10-30% of seminal/vaginal fluids have transmissible virus (Royce et al., 1997; Henin et al., 1993).

In semen viral load, the males HIV-1 infected cells forms about 10^4 of the 10^6 leucocytes per ejaculation (Winkelstein et al., 1987), which confirms AIDS first association with sexual route, with the high prevalence in homosexual men. The virus subsequently became synonymous with heterosexual activity and is now attributed to the AIDS pandemic (UNAIDS 1986; Nkowane 1991; Stoneburner et al., 1990). Bouvier et al (1997) believes that vaginal pH neutralization by semen is a co-factor of HIV transmission.

The chances of transmission also depends on the type of sexually transmitted infections (STI), as co-infection with genital ulcers have been reported to increase the chances of transmission by increasing the susceptibility to HIV infection which also depends on HIV subtypes efficient (Gray et al., 2001; Mahiane et al., 2009; Limpakarnianarat et al., 1993; Wang, 2009; Xu, 2009).

Male circumcision have been documented to decrease the chances of HIV transmission (Mahiane et al., 2009; Lavreys et al., 1999; Gray et al., 2000; Reynolds et al., 2004; Gray et al., 2007; Donoval et al., 2006), but this also depends on the country (Ben et al., 2008; Sullivan et al., 2009; Ruan et al., 2009; Wawer et al., 2009).

The high level of heterosexual spread of HIV in Sub-Saharan Africa and developing countries where genital ulcers from existing venereal diseases (e.g. Chancroid Chlamydia, Syphilis or Herpes virus infections) are aligned with increased HIV seroprevalence (UNAIDS, 1998, Hook et al., 1992; Plummer et al., 1991) could be tight to abrasions at the site of entry in the vagina or anal canal. Heise et al (1991) however reported that HIV could directly infect the bowel mucosa and perhaps cervical epithelium without the need for ulcerations which gave clue to the relatively low risk of the mucosal lining of the foreskin, urethral canal and oral genital contact (through minimal) to be implicated (Winkelstein et al., 1987).

Men having Sex with Men (MSM) have been reported as one of the first way of transmission of HIV. Various authors have showed evidence that the involvement of MSM could be traced to psychosocial behaviour (PB). These PB are said to be depression, violent victimisation, substance abuse, alcohol, psychiatric disorders, psychological distress, lower perceived social support (Berlan et al., 2010; King et al., 2008; Meyer, 2003; Cochran et al., 2003; Cochran and Mays, 2000; Gilman et al., 2001., Marshal et al., 2008; Mimiaga et al., 2009a; b; Safren and Heimberg, 1999; Stall et al., 2001; Chesney et al., 2003; The EXPLORE Study Team, 2004; Herbst et al., 2005). Although some studies have shown how substance use and high risk of HIV transmission are correlated (Stall et al., 2001; Hirshfield et al., 2004), most recent studies are now focussing on how '*syndemic*'- a situation where these diverse psychosocial issues could interact to enhance HIV risky behaviour among MSM (Mustanski et al., 2007; 2010; Stall et al., 2008; Centers for Disease Control and Prevention, 2010). However, varieties of cognitive behavioural interventions have been studied and validated for the treatment of mood and anxiety disorders (Barlow, 2008) behavioural activation therapy and HIV risk reduction counselling in MSM who abuse crystal methamphetamine (Mimiaga et al., 2010).

Addressing co-occurring psychosocial behaviour is a means to increase the effective size of current HIV prevention intervention and allow for more effective uptake by MSM, since they have been reported to be more than 44 times more likely to be newly diagnosed with HIV than other men (Purcell et al., 2010) and the focus on ameliorating disparities in HIV infection is essential for enhancing the health of MSM at the population level (Sanfren et al., 2010).

The Centers for Diseases Control and Prevention (CDC, 2007) reported the prevalence rate among heterosexual African American (AA) women and men with data indicating that more heterosexual AA women having a 74% HIV/AIDS prevalence as compared to the 27% in their male counterpart.

Myths and misperceptions of HIV/AIDS such as HIV being a genocide, suspicion of government information, belief that it is possible to identify risky partners by odour and appearance, belief that partners reported histories are accurate, misperceptions about the meaning of safe sex and the believe that specific classes of people (not one self) are at risk of HIV that resulted from sexual risk contributes to the risky behaviours of HIV transmission (Essien et al., 2002; Catania et al., 1994; Smith et al., 2000; Coleman et al., 2010; Coleman and Ball, 2007; Coleman, 2007).

The increase in the number of sexual partners also increases HIV transmission (Stranford, 1999; Coleman, 2007; Catania et al., 1994; Smith et al., 2000; Coleman et al., 2010; Coleman and Ball, 2007) with most under the influence of alcohol or drugs.

Unprotected oral and vaginal sex have been reported as a risk factor in the transmission of HIV especially where it is carried out in high risk settings, having sex more often under the influence of alcohol and/or drugs (Milam et al., 2006; Catania et al., 1994; Smith et al., 2000).

Even under protection for example the use of condoms, many cases has been reported where the barrier has failed especially where risky behaviours are undertaken. A case in study which made the People Living With HIV/AIDS (PLWHA) in Nigeria to sue the Federal Government of Nigeria to Court for promotion of condoms (Ogunde, 2010).

Though Tenofovir gel has been advocated for women to prevent HIV transmission (Karim et al., 2010).

The nature of HIV transmission from anecdotal records has not changed neither is a new means of transmission of the virus recorded. In view of this development, it is the earnest desire of this write up to bring to fore genealogical reports of the transmission of HIV and to also continue to write on the various modes of transmission as a way of curtailing the spread of the dreaded virus.

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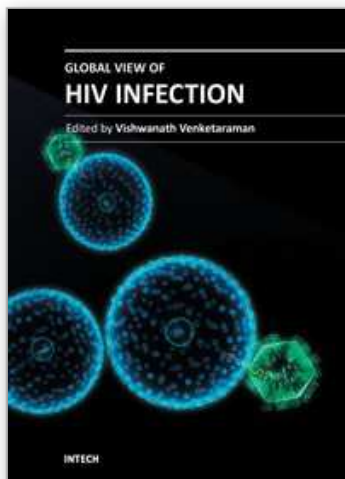
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51000 Rijeka, Croatia
Phone: +385 (51) 770 447
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Unit 405, Office Block, Hotel Equatorial Shanghai
No.65, Yan An Road (West), Shanghai, 200040, China
中国上海市延安西路65号上海国际贵都大饭店办公楼405单元
Phone: +86-21-62489820
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