Syphilis and HIV, HCV and HBsAg co-infections among Sexually Active Adults

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ABSTRACT: The objective of the study was to assess the possibility of syphilis co-infections with human immunodeficiency virus (HIV), hepatitis B virus (HBV), and hepatitis C virus (HCV) among sexually active adults. Screening for HIV, HBV, HCV, and syphilis was carried out by laboratory tests commonly used for diagnosis of HIV, HBV, HCV, and syphilis. Among the 400 samples, serological reactivity was detected for HIV-1/2 in 35(8.8%), HBV in 5(1.3%), HCV in 2(0.5%), and syphilis in 3(0.8%). The incidence of HIV-1/2, HBV and HCV was higher among males 35/179 (19.6%) than females 7/221 (3.2%). Co-infection was observed for HIV—HBV in one (0.3%). None were found to have co-infection with HBV—HCV, HIV—syphilis, HIV—HCV, HBV—syphilis, and HCV—syphilis. Age, sex, locality, and presence of different sexually transmitted infections significantly influence syphilis, HIV, HBV and HCV seropositivity ($P<0.05$). The study shows that a substantial percentage of the samples screened harbor syphilis, HIV and viral hepatitis infections, which otherwise would remain undiagnosed without serological screening.

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1. INTRODUCTION

Sexually transmitted infections (STIs) are a major global cause of infertility, long-term disability and death with severe medical and psychological consequences for millions of men, women and infants (WHO, 2001). The genital ulcerations and inflammation caused by syphilis are implicated as cofactor making infected individuals three to five times more likely to acquire HIV if exposed to the virus through sexual contact. Unless prompt diagnosis and treatment of syphilis are carried out, serious complications including male and female infertility may result, and in pregnancy, adverse outcomes such as still-birth, congenital abnormalities, prenatal death and serious neonatal infection (Schmid, 2004). Patients diagnosed with syphilis should also be tested for other sexually transmitted infections (STIs), including chlamydia, gonorrhea, trichomoniasis, bacterial vaginosis, and HIV infection (Fiumara, 1980; Schmid, 1996; Young, 1998; Augenbraun and Workowski, 1999; Augenbraun and Rolfs, 1999; CDC, 2004; Bai et al., 2008; U.S. Preventive Services Task Force, 2009; Golden and Wasserheit, 2009; Tobian et al., 2009; Hook et al., 2010). According to the Centre for Disease Control and Prevention (CDC) sexually transmitted infections treatment guidelines, pregnant women who are seropositive should be considered infected unless there is evidence of adequate treatment in the medical records and sequential serologic antibody titers have decreased (Workowski and Berman, 2010).

Syphilis, a reportable disease caused by *Treponema pallidum*, is tracked by the Centers for Disease Control and Prevention (CDC). *Treponema pallidum* is a fragile spiral bacterium 6-15 micrometers long by 0.25 micrometers in diameter. Its small size makes it invisible on light microscopy; therefore, it must be identified by its distinctive undulating movements on darkfield microscopy. It can survive only briefly outside of the body; thus, transmission almost always requires direct contact with the infectious lesion (Cox et al., 1992; Fitzgerald, 1992). It can be either acquired or congenital. That is, it can be transmitted either by intimate contact with infectious lesions (most common) or via blood transfusion (if blood has been collected during early syphilis), and it can also be transmitted transplacentally from an infected mother to her fetus (Cox et al., 1992; Fitzgerald, 1992).

Internationally, the prevalence of syphilis varies by region. Syphilis remains prevalent in many developing countries and in some areas of North America, Asia, and Europe, especially Eastern Europe. The highest rates are in South and Southeast Asia, followed closely by sub-Saharan Africa. The third highest rates are in the regions of Latin America and the Caribbean (WHO, 2001). In some regions of Liberia, as of 1999, prevalence was 1300 cases per 100,000 populations (Akovbian et al., 1998). Syphilis is most common during the years of peak sexual activity. Most new cases occur in men and women aged 15-40 years. In 2007, the rate of primary and secondary syphilis was highest in people aged 25-29 years (CDC, 2008).

Men are affected more frequently with primary or secondary syphilis than women. This difference has varied over time. Male-to-female ratios of primary and secondary syphilis increased from 1.6:1 in 1965 to nearly 3:1 in 1985. After, the ratio decreased, reaching a nadir in 1994-95 (CDC, 2008). Males with primary and secondary syphilis outnumber females 6 to 1 (CDC, 2006).

The detection of HBsAg and antibodies to syphilis, HIV, and HCV in plasma or serum is based on the use of rapid enzyme immunoassays (EIAs) or Enzyme Linked Immunosorbent assays (ELISA) which are now commercially available. The specificity of third generation EIAs in the low-prevalence population has been estimated at 99.3 to 100% (Vrielink et al., 1997). Falsely positive results can, however be observed due to cross-reactivity with other viral antigens or immunological disorders (Essex et al., 1990; Kovacs et al., 1995). The presence of HBsAg in serum or plasma is an indication of active Hepatitis B infection, either acute or chronic. Similarly, anti-bodies to hepatitis C virus (anti-HCV) are used to detect HCV infection (Olokoba et al., 2008, 2009). The *in-vitro* detection of these antibodies using synthesized HIV-1/2 peptides has been used as diagnostic tool. The least expensive means of knowing one's HIV serostatus is by testing human blood samples for the presence of HIV-1/2 specific antibodies (WHO, 2004). Such testing has been used for surveillance study of HIV infection globally which provides invaluable epidemiological data for health planning/ programme regarding HIV and AIDS (WHO, 2004). ELISAs, western Blots, PCR-based assays and various other test systems are currently available for HIV-1/2 detection (Essex et al., 1990; Kovacs et al., 1995). In this regard, rapid HIV serodiagnosis which is less costly, with reduced turnaround time is especially suitable.

Infections with HIV, HBV, and HCV are major public health problems (Hussain et al., 2006). Many risk behaviors as well as the routes of transmission for HBV and HCV infections are identical to those for HIV and other sexually transmitted infections (STIs) (Hussain et al., 2006). Early diagnosis and effective treatment of STDs, especially those that cause ulcers and blood-borne infections, are an important strategy for the prevention of HIV transmission (Hussain et al., 2006).

This study was carried out to assess the possibility of *Treponema pallidum* co-infections with HIV, HBV, and HCV among sexually active adults.

2.0. MATERIALS AND METHODS

2.1. STUDY AREA

The study area is the Blood Grouping & Serology Unit, University College Hospital (UCH), and the Lead City University Medical Centre, both located at the municipal area of Ibadan, which is made up of five local government areas. Ibadan is the capital city of Oyo State located in the forest zone of southwestern Nigeria. Ibadan city lies on the longitude 3°5' East of Greenwich meridian and latitude 7°23' North of the Equator. Besides being the largest indigenous city in Africa south of Sahara, the city is an important trade and educational centre. It also houses one of the largest and foremost teaching hospitals in Africa. However, the city is characterized by low level of environmental sanitation, poor housing, and lack of potable water and improper management of wastes especially in the indigenous core areas characterized by high density and low income populations.

2.2. STUDY POPULATION

Two hundred samples were collected from at the Blood Grouping & Serology Unit, University College Hospital, Ibadan, South-Western, Nigeria for serological analysis. Also, two hundred (200) patients (10 males and 190 females) of different ages and socioeconomic status, who attended the STI clinic of LCU Medical Centre in Ibadan, with one or more of the complaints as enunciated by WHO in its syndromic approach for the diagnosis of STI (WHO, 1991; Choudhry et al., 2010) were included as subjects. All samples were screened for syphilis and viral STIs by standard microbiological methods (Collee et al., 1989; Cheesbrough, 2006; Choudhry et al., 2010). Permission and consent were obtained before

carrying out the study. Table 1 shows demographic profiles of the subjects under study comprising of sexually active attendees of Lead City University Medical Centre, Ibadan, Southwestern Nigeria and the blood donors at UCH, Ibadan.

Table 1: Demographic profiles of the subjects used in this study

Profiles	No. Tested (%)	No. UCH (%)	No. LCU (%)
Age Group (years)			
16-39	340(85.0)	152(44.7)	188(55.3)
40 and above	60(15.0)	48(80.0)	12(20.0)
Sex			
Males	179(44.8)	169(94.4)	10(5.6)
Females	221(55.2)	31(14.0)	190(86.0)
Marital status			
Married	83(20.8)	73(88.0)	10(12.0)
Single	317(79.2)	127(40.1)	190(59.9)
Total	**400(100.0)**	**200(50.0)**	**200(50.0)**

2.3. SAMPLE COLLECTION AND PREPARATION

The method of sample collection employed was venepuncture technique (Cheesbrough, 2006). About 3 ml of blood was collected and transferred into an EDTA bottle. This was centrifuged and the plasma was then pipetted into sterile ependorf tubes and stored at -20^{0}C until ready for use.

2.4. ASSAY FOR TREPONEMA PALLIDUM SPECIFIC ANTIBODIES

Each serum sample was screened for *T. pallidum* specific antibodies at room temperature using The Syphilis Ultra Rapid Test Strip (Whole Blood/Serum/ Plasma) [ACON(R) Laboratories Incorporated USA Lot No: SYP 70800H5 and Global Device, USA]. The Syphilis Ultra Rapid Test Strip (Whole Blood/Serum/ Plasma) is a rapid chromatographic immunoassay for the qualitative detection of antibodies (IgG and IgM) to *Treponema Pallidum* (TP) in whole blood, serum or plasma to aid in the diagnosis of Syphilis. The results were interpreted according to the manufacturer's specifications. The Syphilis Ultra Rapid Test Strip (Whole Blood/Serum/Plasma) has a relative sensitivity of 99.7% and relative specificity of 99.6%.

2.5. SEROLOGICAL ANALYSIS OF HIV, HCV AND HBsAg

Separated serum from blood samples were dispensed into two 3 ml volumes sterile plastic containers and used within two days for screening tests of HIV, HCV, and Hepatitis B surface Antigen (HBsAg). Others were frozen for confirmatory tests. These viruses were screened using parallel rapid test kits. HIV antibody assay was carried out with Determine HIV 1/2 rapid test strips (Abbott laboratories-USA) and HIV 1/2 Stat-Pak assay (Chembio diagnostics – USA) methods according to the standard national HIV screening algorithm in Nigeria (FMoH, 2005). These tests are qualitative membrane-based immuno assay techniques. Hepatitis B surface antigen test was done using Hepatitis B surface antigen test strips (IND^R Diagnostica, Canada and Global Diagnostics, USA). Hepatitis C antibody was tested using HCV-Ab test strips (IND^R Diagnostica, Canada and Global Diagnostics, USA).

2.6. DATA ANALYSIS

The data generated in this study were analyzed at 5% level of significance by Chi-square statistical test using SPSS 13.0 for windows. Data was presented using descriptive statistics for syphilis, HIV, HCV and HBsAg.

3.0. RESULTS ANALYSIS

The results of 400 samples screened are shown in Table 2-3.

3.1. Syphilis Status with other co-infections among subjects used in this study

Tables 2-3 shows that of the 400 subjects, 35(8.8%) were HIV positive, 5(1.3%) were positive for hepatitis B virus by rapid assays, 2(0.5%) were positive for antibodies to the hepatitis C virus by rapid assays, 3(0.8%) had antibodies to syphilis (reactive for syphilis by VDRL test) and 1(0.3%) was positive for HIV & HBV co-infections by rapid assays (Tables 2-3).

3.2. Prevalence of HIV, HBV, HCV, and syphilis co-infections status of subjects used in this study in relation to sex

Table 2 shows prevalence of HIV, HBV, HCV, and syphilis co-infections status of subjects used in this study in relation to sex. Of the 400 subjects, 2(0.9%) females and 1(0.6%) male were infected with *Treponema pallidum* (syphilis). Also, of the 400 subjects, 29(16.2%) males and 6(2.7%) females were infected with HIV and 4(2.2%) males and 1(0.5%) female were infected with HBV and only 2(1.1%) males were infected with HCV. HIV-HBV co-infections were observed in 1(0.6%) male subjects. More males [35(19.6%)] than females [7(3.2%)] had viral STIs. The difference in syphilis positive, HBV, HCV and HIV positive status was significant ($p< 0.05$) between male and female subjects.

Table 2: HIV, HBV, HCV, and syphilis co-infections status of subjects used in this study in relation to sex

Subjects		No. Tested (%) n= 400	Males (%) n= 179	Females (%) n= 221
HIV Status	Reactive	35(8.8)	29(16.2)	6(2.7)
	Non-reactive	365(91.2)	150(83.8)	215(97.3)
HCV Status	Reactive	2(0.5)	2(1.1)	0(0.0)
	Non-reactive	398(99.5)	177(98.9)	221(100.0)
HBV Status	Reactive	5(1.3)	4(2.2)	1(0.5)
	Non-reactive	395(98.7)	175(97.8)	220(99.5)
Syphilis Status	Reactive	3(0.8)	1(0.6)	2(0.9)
	Non-reactive	397(99.2)	178(99.4)	219(99.9)
HIV-HBV	Reactive	1(0.3)	1(0.6)	0(0.0)
	Non-reactive	399(99.7)	178(99.4)	221(100.0)
HIV-HCV	Reactive	0(0.0)	0(0.0)	0(0.0)
	Non-reactive	400(100.0)	179(100.0)	221(100.0)
HIV-Syphilis	Reactive	0(0.0)	0(0.0)	0(0.0)
	Non-reactive	400(100.0)	179(100.0)	221(100.0)
HIV-HBV-HCV-Syphilis	Reactive	0(0.0)	0(0.0)	0(0.0)
	Non-reactive	400(100.0)	179(100.0)	221(100.0)
HBV-HCV	Reactive	0(0.0)	0(0.0)	0(0.0)
	Non-reactive	400(100.0)	179(100.0)	221(100.0)
Total for Viral STIs		**42(10.5)**	**35(19.6)**	**7(3.2)**

Table 3: Syphilis and HIV Status among subjects used in this study in relation to their demographic profiles

Profiles		No. Tested (%) (n=400)		Males (%) (n=179)		Females (%) (n=221)	
Syphilis Status		**No. Tested**	**Syphilis Positive**	**No. Tested**	**Syphilis Positive**	**No. Tested**	**Syphilis Positive**
Age group	16-39 years	340(85.0)	2(0.6)	138(40.6)	1(0.7)	202(59.4)	1(0.5)
	40 yrs & above	60(15.0)	1(1.7)	44(73.3)	0(0.0)	16(26.7)	1(6.3)
Sex	Males	179(44.7)	1(0.6)	179(100.0)	1(0.6)	0(0.0)	0(0.0)
	Females	221(55.3)	2(0.9)	0(0.0)	0(0.0)	221(100.0)	2(0.9)
Marital status	Married	83(20.7)	1(1.2)	56(67.5)	0(0.0)	27(32.5)	1(3.7)
	Single	317(79.3)	2(0.6)	123(38.8)	1(0.8)	194(61.2)	1(0.5)
Location	UCH	200(50.0)	0(0.0)	169(84.5)	0(0.0)	31(15.5)	0(0.0)
	LCU	200(50.0)	3(1.5)	10(5.0)	1(10.0)	190(95.0)	2(1.0)
Total		**400(100.0)**	**3(0.8)**	**179(44.8)**	**1(0.3)**	**221(55.2)**	**2(0.5)**
HIV Status		**No. Tested**	**HIV Positive**	**No. Tested**	**HIV Positive**	**No. Tested**	**HIV Positive**
Age group	16-39 years	340(85.0)	28(8.2)	138(40.6)	23(16.7)	202(59.4)	5(2.5)
	40 yrs & above	60(15.0)	7(11.7)	44(73.3)	6(13.6)	16(26.7)	1(6.3)
Sex	Males	179(44.7)	29(16.2)	179(100.0)	29(16.2)	0(0.0)	0(0.0)
	Females	221(55.3)	6(2.7)	0(0.0)	0(0.0)	221(100.0)	6(2.7)
Marital status	Married	83(20.7)	14(16.9)	56(67.5)	10(17.9)	27(32.5)	4(14.8)
	Single	317(79.3)	21(6.6)	123(38.8)	19(15.4)	194(61.2)	2(1.0)
Total		**400(100.0)**	**35(8.8)**	**179(44.8)**	**29(7.3)**	**221(55.3)**	**6(1.5)**
HBV Status		**No. Tested**	**HBsAg**	**No. Tested**	**HBsAg Positive**	**No. Tested**	**HBsAg Positive**
Age group	16-39 years	340(85.0)	3(0.9)	138(40.6)	2(0.7)	202(59.4)	1(0.5)
	40 yrs & above	60(15.0)	2(3.3)	44(73.3)	2(0.0)	16(26.7)	0(0.0)
Sex	Males	179(44.7)	4(2.2)	179(100.0)	4(0.6)	0(0.0)	0(0.0)
	Females	221(55.3)	1(0.5)	0(0.0)	0(0.0)	221(100.0)	1(0.9)
Marital status	Married	83(20.7)	3(3.6)	56(67.5)	2(0.0)	27(32.5)	1(3.7)
	Single	317(79.3)	2(0.6)	123(38.8)	2(0.8)	194(61.2)	0(0.0)
Total		**400(100.0)**	**5(1.3)**	**179(44.8)**	**4(1.0)**	**221(55.3)**	**1(0.3)**
HCV Status		**No. Tested**	**HCV Positive**	**No. Tested**	**HCV Positive**	**No. Tested**	**HCV Positive**
Age group	16-39 years	340(85.0)	1(0.3)	138(40.6)	1(0.7)	202(59.4)	0(0.0)
	40 yrs & above	60(15.0)	1(1.7)	44(73.3)	1(2.3)	16(26.7)	0(0.0)
Sex	Males	179(44.7)	2(1.1)	179(100.0)	2(1.1)	0(0.0)	0(0.0)
	Females	221(55.3)	0(2.7)	0(0.0)	0(0.0)	221(100.0)	0(0.0)
Marital status	Married	83(20.7)	1(1.2)	56(67.5)	1(1.8)	27(32.5)	0(0.0)
	Single	317(79.3)	1(0.3)	123(38.8)	1(0.8)	194(61.2)	0(0.0)
Total		**400(100.0)**	**2(0.5)**	**179(44.8)**	**2(0.5)**	**221(55.3)**	**0(0.0)**

3.3. Syphilis and HIV Status among subjects used in this study in relation to their demographic profiles

Table 3 shows the socio-demographic characteristics, HIV status among subjects under study. Of 400 patients, 35 (8.8%) were HIV positive and 3(0.8%) were reactive for syphilis. This suggests that HIV affect a large percentage of clinic patients. Of the 400 subjects, 2(0.6%) subjects in the 16—39 years age group and 1(1.7%) in the 40 years and above age group were HIV positive. The difference in syphilis positive status was significant (P<0.05) between ages of subjects. Their marital status showed that 1(1.2%) married subjects and 2(0.6%) singles were infected with syphilis. However, the difference in syphilis positive status was significant (P<0.05) between the singles and married status of the subjects under study. Also among the 400 subjects, 28(8.2%) subjects in the 16—39 years age group and seven (11.7%) in the 40 years and above age group were HIV positive. The difference in HIV positive status was not significant (P<0.05) between ages of subjects. Their marital status showed that 14(16.9%) married subjects and 21(6.6%) singles were infected with HIV. However, the difference in HIV positive status was significant (P<0.05) between the singles and married status of the subjects under study. Their educational status, occupations and clinical history was not defined. The results revealed that the variables— sex, age, marital status, and locality — were found to significantly influence syphilis and HIV positivity (P<0.05).

4.0. DISCUSSION

In this study, the overall prevalence rate was 8.8% for HIV, 1.3% for HBV, 0.5% for HCV, 0.8% for syphilis and 0.3% for HIV/HBV co-infection. This study showed a high prevalence rate for HIV among the subjects under study. It is higher than the <4.1% reported for Oyo State in the last national sentinel survey. Nigeria's epidemic is characterized as one of the most rapidly increasing rates of HIV and AIDS. The prevalence rate of 8.8% recorded for HIV in this study deviates from that of the Federal Ministry of Health (2005) Sentinel Study on HIV in Nigeria. Elsewhere, higher seroprevalence rate among different populations have been reported (FMoH, 2005; Pennap et al., 2006; Motayo et al., 2009; Akinjogunla and Adegoke, 2009). The prevalence of viral infection also varied with age, the highest prevalence rate was recorded in age group 40 years and above. This agrees with previous findings, Akinjogunla and Adegoke (2009) reported a significant difference in the age of the individuals with the viral infection and the prevalence of the viral infection in relation to age to be 13.6% (n=43) for age group 31-45 years.

VDRL test reactive strip samples found significant treponemal antibodies in 3 of them, absence of significant treponemal antibodies in other samples tested with VDRL strips may suggest a non syphilitic reagin antibody production or cross reactions with endemic treponemal infections such as yaws, (*T. pertenue*), pinta (*T. carateum*) or bejel (*T. endemicum*) (Noris, 2003). The detection of these positives clearly reveals the non specificity of the rapid test strips and the need for a specific confirmatory test for syphilis (Larsen, 1995), especially as this is not the practice in most hospitals.

The prevalence rate of 0.5% recorded for HCV in this study deviates from that of the Udeze et al. (2011), who reported a prevalence of 8.0% among first year students of Univeristy of Ilorin in Nigeria. This prevalence is lower than 3% worldwide seroprevalence reported by the World Health Organization in 1999 (WHO, 1999). Elsewhere, higher seroprevalence rate of HCV among different populations have been reported (Halim and Ajayi, 2000; Inyama *et al*., 2005; Ogunro *et al*., 2007; Ndako *et al*., 2009; Udeze *et al.,* 2009). Oni and Harrison (1996) earlier reported a lower prevalence of 2.0% among the general population in Nigeria. Similarly, a lower prevalence of 1.2% was recorded among general population in Tanzania (Tess *et al*., 2000). Factors that have been reported to influence the rate of HCV disease progression include age (increasing age associated with more rapid progression), gender (males have more rapid disease progression than females), alcohol consumption (associated with an increased rate of disease progression) (Robert, 2009). HIV co infection (associated with a markedly increased rate of disease progression), fatty liver (the presence of fat in liver cells has been associated with an increased rate of disease progression) (Karmochkine *et al*., 2006), and diabetes (Ndako *et al*., 2009).

Analysis of the results revealed that the variables—sex, age, marital status, and locality — significantly influenced the rate of syphilis, HIV, HBV and HCV seropositivity among the population under study. This agrees with what has been previously reported by some authors (Hussain et al., 2006). More males [35(19.6%)] than females [7(3.2%)] had viral STIs, but this could be due to the larger number of females, 221 as compared to 179 males screened during the study. With regards to HIV, more males

[29(16.2%)] were affected by HIV than females [6(2.7%)]. This disagrees with what was reported by Hussain et al. (2006). Akinjogunla and Adegoke (2009) reported a significant difference in the sex of the individuals with the viral infection and the prevalence of the viral infection in relation to sex to be 39.2% for males and 31.8% for females respectively.

Also in this study, more females, i.e., 2(0.9%) had syphilis than males, 2(0.6%) but this could be due to the larger number of females, 221 as compared to 179 males screened during the surveillance. With regard to HIV-HBV co-infection, males 1(0.3%) were only affected than females. More males, i.e., 4(2.2%) had HBV than females, 1(0.5%). Only males, i.e. 2(1.1%) had HCV in this study. This result disagrees with the finding of Ndako *et al.* (2009) who reported higher prevalence in diabetic female patients in Jos, Nigeria; Ejele *et al*. (2006) who reported that females had higher HCV antibodies prevalence than males in Niger Delta, Nigeria and Udeze et al. (2011) who also reported that females had higher HCV antibodies prevalence than males in Ilorin, Nigeria. This result however agrees with the report of Baba *et al*. (1998) that prevalence of viral hepatitis is higher among the males in Nigeria.

Men are affected more frequently with primary or secondary syphilis than women. This difference has varied over time. Male-to-female ratios of primary and secondary syphilis increased from 1.6:1 in 1965 to nearly 3:1 in 1985. After, the ratio decreased, reaching a nadir in 1994-95 (CDC, 2008). Since 2002, the incidence of primary and secondary syphilis has risen 54% among men. Among women, the rates of primary and secondary syphilis remain lower. After a decade of declines, the overall prevalence of syphilis among females increased 11.1% between 2005 and 2006. Males with primary and secondary syphilis outnumber females 6 to 1 (CDC, 2006). The recent increase in the male-to-female ratio is largely attributable to the increased rate of disease among men having sex with men (MSM) (CDC, 2008). Studies of patients diagnosed with sexually transmitted diseases (STDs) demonstrate that men are screened for syphilis in emergency departments and health clinics more often than women. Although surveillance data based on risk behavior are not available, a separate CDC analysis suggests that approximately 64% of all adult primary and secondary syphilis cases in 2004 were among MSM, up from an estimated 5% in 1999. In 2007, 65% of new cases occurred in MSM, and there is a high rate of HIV co-infection (CDC, 2008).

There was only HIV-HBV co-infections in one (0.3%) of the subjects. No co-infections of HIV—HCV, HBV—HCV, HIV—syphilis, and HIV-HBV-HCV-syphilis infections. Although the percentage of patients with co-infections is zero, the combination of viral infections such as HIV and HBV or HBV and HCV is a dangerous co-existence (Thio et al., 2002; Mosunjac et al., 2003; Ramia et al., 2003) and may have a detrimental effect on the patient and the treatment outcome. None were found to be co-infected with HIV—HCV, HBV—syphilis, or HCV—syphilis. This agrees favourably with what was reported by Hussain et al. (2006).

5.0. CONCLUSION

The results reveal that sexually active hospital attendees in Ibadan harbor blood-borne viral infections like HIV, HBV, and HCV, which would otherwise remain undiagnosed in the absence of screening. Further, they are unaware of the underlying co-infection because this was an unlinked anonymous testing of coded samples (Hussain et al., 2006). Screening the high-risk population for these viral infections would aid early detection of co-infections and hence early treatment, which, if initiated, would help to decrease the further spread of these blood-borne infections. There is a need, therefore, to support an approach of targeted screening of all these viral infections, integrating viral hepatitis testing, counseling and referral services into the existing STI prevention and treatment services.

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REFERENCES

1. Akinjogunla OJ and Adegoke AA. Sero-prevalence of human immunodeficiency virus (HIV) 1 and 2 infections in Uyo metropolis, Akwa Ibom State. *Scientific Research and Essay,* 2009; *4 (11)*:1381-1384

2. Akovbian VA, Gomberg MA, Prokhorenkov VI. Syphilitic vignettes from Russia. *Dermatol Clin*. Oct 1998;16(4):687-90, x.
3. Augenbraun M, Workowski K. Ceftriaxone therapy for syphilis: report from the emerging infections network. *Clin Infect Dis*. Nov 1999;29(5):1337-8.
4. Augenbraun MH, Rolfs R. Treatment of syphilis, 1998: nonpregnant adults. *Clin Infect Dis*. Jan 1999;28 Suppl 1:S21-8.
5. Baba MM, Gashau W and Hassan AW. Detection of Hepatitis-B surface Antigenaemia in Patients with and Without the Manifestations of AIDS in Maiduguri, Nigeria. *The Nigerian Postgraduate Medical Journal*, 1998; 5:125-128.
6. Bai ZG, Yang KH, Liu YL, Tian JH, Ma B, Mi DH, et al. Azithromycin vs. benzathine penicillin G for early syphilis: a meta-analysis of randomized clinical trials. *Int J STD AIDS*. Apr 2008;19(4):217-21.
7. Center for Disease Control & Prevention (CDC&P)(2006). Sexually Transmitted Diseases Treatment Guidelines. Morbidity Mortality Weekly Recommendation Report, 55 (RR-11): www.cdc/gov/std/treatment.
8. Center for Disease Control (CDC, 2008). National STD prevention conference. Confronting challenges, applying solutions. Division of STD prevention for HIV/AIDS Viral Hepatitis, STD and TB prevention. CDC Press Release. (404): 639-889.
9. Centers for Disease Control and Prevention (CDC). Azithromycin treatment failures in syphilis infections--San Francisco, California, 2002-2003. *MMWR Morb Mortal Wkly Rep*. Mar 12 2004;53(9):197-8.
10. Cheesbrough M. 2006. Medical Laboratory Manual for Tropical Countries ELBS 7: 312 - 315.
11. Choudhry S, Ramachandran VG, Das S, Bhattacharya SN, Mogha NS. Pattern of sexually transmitted infections and performance of syndromic management against etiological diagnosis in patients attending the sexually transmitted infection clinic of a tertiary care hospital. Indian J Sex Transm Dis 2010;31:104-8
12. Choy Y, Gittens-Williams L, Apuzzio J, Skurnick J, Zollcoffer C, McGovern PG. Risk factors for hepatitis C infection among sexually transmitted disease-infected, inner city obstetric patients. Infect Dis Obstet Gynecol 2003;11:191—198.
13. Collee TG, Duguid JP, Fraser AG, Marmion BP, editors. Mackie and Mc Cartney Practical Medical Microbiology 14 thed. New York: Churchill Livingstone; 1989.
14. Cox DL, Chang P, McDowall AW, Radolf JD. The outer membrane, not a coat of host proteins, limits antigenicity of virulent Treponema pallidum. *Infect Immun*. Mar 1992;60(3):1076-83.
15. Essex M, Kanki PJ, Marlink R, et al. (1990). Antigenic characterization of the human immunodeficiency viruses. *Journal of American Academy of Dermatology 22*: 1206-1210
16. Ejele, O. A., Nwauche, C. A. and Erhabor, O. (2006). Seroprevalence of Hepatitis C Virus in the Niger Delta of Nigeria. *The Nigerian Postgraduate Medical Journal*, 13(2): 103-106.
17. Federal Ministry of Health (FMoH) (2005). Technical Report on National HIV/Syphilis Sero-Prevalence Sentinel Survey Among Pregnant Women Attending Antenatal Clinics in Nigeria.
18. Fitzgerald TJ. The Th1/Th2-like switch in syphilitic infection: is it detrimental?. *Infect Immun*. Sep 1992;60(9):3475-9.
19. Fiumara NJ. Treatment of primary and secondary syphilis. Serological response. *JAMA*. Jun 27 1980;243(24):2500-2.
20. Golden MR, Wasserheit JN. Prevention of viral sexually transmitted infections--foreskin at the forefront. *N Engl J Med*. Mar 26 2009;360(13):1349-51.
21. Halim, N. K. and Ajayi, O. I. (2000). Risk factors and Seroprevalence of Hepatitis C Antibody in Blood Donors in Nigeria. *East African Medical Journal*, 77: 410-412.
22. Hook EW 3rd, Behets F, Van Damme K, Ravelomanana N, Leone P, Sena AC, et al. A phase III equivalence trial of azithromycin versus benzathine penicillin for treatment of early syphilis. *J Infect Dis*. Jun 1 2010;201(11):1729-35.
23. Hussain T, Kulshreshtha KK, Sinha S, Yadav VS, Katoch VM. (2006). HIV, HBV, HCV, and syphilis co-infections among patients attending the STD clinics of district hospitals in Northern India. International Journal of Infectious Diseases, 10:358—363
24. Inyama, P. U., Uneke, C. J. Anyanwu, G. I. Njoku, O. M. Idoko, J. H. and Idoko, J. A. (2005). Prevalence of Antibodies to Hepatitis C virus among Nigerian Patients with HIV Infection. *Online J. Health and Allied Sci*., 2:2.www.ojhas.org/issue14/2005-2-2.htm. Retrieved 2009April, 12
25. Karmochkine, M., Carrat, F., Dos Santos, O., Cacoub, P. and Raguin, G. (2006). A Case-Control Study of Risk Factors for Hepatitis C Infection in Patients with unexplained Routes of Infection. *Journal of Viral Hepatology*, 13 (11): 775–782.

26. Kovacs A, Xu J, Rasheed S, et al. (1995). Comparison of a rapid non-isotopic polymerase chain reaction assay with four commonly used methods for the study of human immunodeficiency virus type 1 infection in neonates and children. *Pediatrics Infectious Diseases Journal 14*: 948-954
27. Larsen, S. A. (1995). Laboratory diagnosis and interpretation of tests for syphilis. Clinical Microbiology Reviews, (130) 2353 – 2358.
28. Mosunjac MB, Tadros T, Beach R, Majumdar B. Cervical schistosomiasis, human papillomavirus (HPV), and human immunodeficiency virus (HIV): a dangerous co-existence or co-incidence? Gynecol Oncol 2003;90:211—4.
29. Motayo OB, Okonko IO, Uche LN, Onoja BA, Obiogbolu CH. 2009. The Sero-prevalence of Human Immunodeficiency Virus (HIV) among Patients Attending the Special Treatment Clinic at University College Hospital, Ibadan, Nigeria. *Nigerian Journal of Microbiology 23(1):* 1904-1910
30. Nanu A, Sharma SP, Chatterjee K, Jyoti P. Markers for transfusion transmissible infections in north Indian voluntary and replacement blood donors: prevalence and trends 1989—1996. Vox Sang 1997;73:70—3
31. Ndako, J. A., Echewu, G. O., Shidali, N. N., Bichi, I. A., Paul, G. A., Onovoh, E. and Okeke, L. A. (2009). Occurrence of Hepatitis C Virus Infection in type 2 Diabetic Patients Attending Plateau State Specialist Hospital Jos, Nigeria. *Virology Journal,* 6**:**98doi:10.1186/1743-422X-6-98.
32. Noris, S. J. (2003). Treponema and other human host- associated spirochetes. In: Manual of Clinical Microbiology, 8th edition Murray P. R et al.,(Editors). Amsterdam Society of Medicine Press.
33. Ogunro PS, Adekanle DA, Fadero FF, Ogungbamigbe TO, Oninla SO. (2007). Prevalence of anti-hepatitis C virus antibodies in pregnant women and their offspring in a tertiary hospital in Southwestern Nigeria. *J. Infect Developing Countries; 1(3)*: 333-336.
34. Olokoba AB, Olokoba LB, Salawu FK, Danburam A, Desalu OO, Midala J, Aderibigbe S. Hepatitis C virus and Human immunodeficiency virus co-infection in North-Eastern Nigeria. *Research Journal of Medical Sciences*, 2008; *2(5)*:217-219.
35. Olokoba AB, Salawu FK, Danburam A, Desalu OO, Olokoba LB, Wahab KW, Badung LH, Tidi SK, Midala J, Aderibigbe S, Abdulrahman MB, Babalola OM and Abdukkarim A. Viral Hepatitides in Voluntary Blood Donors in Yola, Nigeria. European Journal of Scientific Research, 2009; 31(3):329-334
36. Oni, A. O. and Harrison, T. J. (1996). Genotypes of Hepatitis C Virus in Nigeria. *Journal of Medical Virology*, 49: 178-186.
37. Pennap GRI, Makut MD, Gyar SD, Owuna G (2006). Sero-prevalence of HIV/AIDS in Keffi and Environs. Niger. J. Microbiol. 20(3):1114-1146.
38. Ramia S, Klayme S, Naman R. Infection with hepatitis B and C viruses and human retroviruses (HTLV-I and HIV) among high-risk Lebanese patients. Ann Trop Med Parasitol 2003;97: 187—92.
39. Roberts, W. B. (2009). *Microbiology with Disease by Body system*. 2nd Edition. Pearson Education inc. Sanfrancisco. pp714-716.
40. Schmid G. 2004. Economic and programmatic aspects of congenital syphilis prevention. Bull of World Health Organization 82: 4-02 4-09.
41. Schmid GP. Serologic screening for syphilis. Rationale, cost, and realpolitik. *Sex Transm Dis*. Jan-Feb 1996;23(1):45-50.
42. Tess, B. H., Levin, A., Brubaker, G., Shao, J., Drummond, J. E., Alter, H. J. and O'Brien, T. R. (2000). Seroprevalence of Hepatitis C Virus in the General population of Northeast Tanzania. *American journal of Tropical Medicine*, 62(1):138-141.
43. Thio CL, Seaberg EC, Skolasky Jr R, Phair J, Visscher B, Munoz A, et al. HIV-1, hepatitis B virus, and risk of liver-related mortality in the Multi-centre Cohort Study (MACS). Lancet 2002;360: 1921—6.
44. Tobian AA, Serwadda D, Quinn TC, Kigozi G, Gravitt PE, Laeyendecker O, et al. Male circumcision for the prevention of HSV-2 and HPV infections and syphilis. *N Engl J Med*. Mar 26 2009;360(13):1298-309.
45. Udeze, A. O., Okonko, I. O., Donbraye, E., Sule, W. F., Fadeyi, A. and Uche, L. N. (2009). Seroprevalence of Hepatitis C Virus Antibodies Amongst Blood Donors in Ibadan, Southwestern, Nigeria. *World Applied sciences Journal*, 7(8): 1023 – 1028.
46. Udeze AO, Bamidele RA, Okonko IO, Sule WF.2011. Hepatitis C Virus (HCV) Antibody Detection among First Year Students of University of Ilorin, Ilorin, Nigeria. *World Journal of Medical Sciences 6(3): 162-167*
47. United States Preventive Services Task Force. Screening for syphilis infection in pregnancy: U.S. Preventive Services Task Force reaffirmation recommendation statement. *Ann Intern Med*. May 19 2009;150(10):705-9.
48. Vrielink, H., Reesink, H.W., Vandon Burg, P. J., Laaijer, H. L., Crypers, H. T., lilie, P. N.,

Vander Poel, C. L., Performance of three generations of anti-hepatitis C virus enzyme-linked immunosorbent assay in donors and patients. Transfusion, 1997; 37(8): 845-849.
49. Workowski KA, Berman S. CDC Sexually transmitted diseases treatment guidelines, 2010. *MMWR Recomm Rep*. Dec 17 2010;59:1-110.
50. World Health Organization (WHO) (2005). Guidelines for the Treatment of Sexually Transmitted Diseases. Morbidity Mortality Weekly Recommendation Report, 47 (RR-1); 1-118.
51. World Health Organization (WHO). 2001. Global prevalence and incidence of selected curable sexually transmitted infections: Overview and estimates. Geneva: 2001.
52. World Health Organization. (1999). Global Surveillance and Control of Hepatitis C. Report of a WHO consultation organized in collaboration with the Viral Hepatitis Prevention Board, Antwerp, Belgium. *Journal of Viral Hepat.* 6: 35-47.
53. World Health Organization. Management of patients with sexually transmitted diseases. WHO Technical Report Series No. 810. Geneva: World Health Organization; 1991
54. Young H. Syphilis. Serology. *Dermatol Clin*. Oct 1998;16(4):691-8.

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