INCIDENCE OF HIV INFECTIONS AMONGST PATIENTS WITH NEISSERIA GONORRHOEAE ATTENDING SEXUALLY TRANSMITTED DISEASES CLINIC, AHMADU BELLO UNIVERSITY TEACHING HOSPITAL ZARIA

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DECLARATION

I, hereby solemnly declare that the work reported in this thesis was carried out by me in the department of microbiology, Ahmadu Bello University, Zaria under the supervision of Prof. (Mrs) L. J. Ogbadu and Dr. (Mrs) O. Y. Elegba.

No part of this work has been previously submitted for a degree, diploma or qualification before. The work of other investigators in this area is duly acknowledged and referred to appropriately.

15/9/97

DATE

ILIYA SANI

DEDICATION

This work is dedicated to my parents Mallam Iliyasu Sule and Madam Maimunat Iliyasu, My wife Sa'adat, My Brothers Rabi'u and Kamisu, Sisters, Suwaiba, Akalima, Shata'atu, Magajiya, Hadiza, Safiya and Karimatu.

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ABSTRACT

Two hundred patients consisting of one hundred and fifty eight males and forty two females were screened for gonococeal infections. Their ages ranged from 15 50 years. Microbiological diagnosis was from urethral and endocervical/high vaginal swabs (HVS) using wet-film preparation, gram stain technique and culture on chocolate agar. Blood samples were obtained for HIV testing from the. confirmed cases of gonorrhea patients. HIV serologic testing was performed by enzyme linked immunosorbent assay (Wellcozyme 1 and 2). Infection with Neisseria gonorrheae was demonstrated in 25 (12.5) of the sample tested in which 17 (68%) were males and 8 (32%) were females. HIV seropositivity accounted for 16 % (4/25) among patients with positive cultures. More men with gonococcal infections were found to be HIV seropositive 3 (17.6%) as compared to their female counterparts 1 (12.5%). Apart from Neisseria gonorrheae there were other STDs isolated amongst the patients (analysed in the) studied. These included Candida sp 11(5.5%) and staphylococcus sp 45 (22.5%). Antibiotic susceptibility pattern showed high resistance to penicillin and ampicillin, sensitive to only 12% and 16% and resistant to 88% and 84% respectively. The most effective antiobiotic against Neisseria gonorrheae was ofloxacin with 100% sensitivity followed by ceftriazone, gentamycin, erythromycin, tetracycline and cefuroxime all of which showed over 60% sensitivity.

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CHAPTER ONE

INTRODUCTION

Gonorrheae is a sexually transmitted disease caused by Neisseria gonorrheae, a gram negative diplococcus. The disease affects both men and women. In males and females, gonorrhea has been endemic throughout the world for many centuries and from earliest history to modern days, gonococcal urethritis has continued to be a problem of serious impact. According to the centre for disease control (CDC) bulletin (1992), there were 586,638 reported cases of gonorrhea in USA in 1991 alone.

In men, gonorrheae usually presents with purulent discharge from the urethra, painful and frequency of urination (Jawetz et al., 1984). In females, gonorrhea infections present with abnormal vaginal discharge, prolonged or abnormal menstruation and painful urination. Sometimes the infection may remain symptomless in females (Stokes, 1987). Untreated gonorrhea in man may lead to epididymitis, proctitis, urethral stricture, sterility and infertility. Females may develop complications such as pelvic inflammatory disease (PID), chronic abdominal pain, ectopic pregnancies and counsequent sterility (William, 1984).

When the prevalence figures of some African countries are compared with those of the developed countries, it is obvious that sexually transmitted diseases in Africa constitute a major public health problem. For example, the incidence of gonorrhea per 100,000 population in Lagos (Nigeria) is 49,076,

Kampala (Uganda) is 10,000 and in Nairobi (Kenya) it is 7,000; the corresponding figures for London (Britain) and Atlanta (USA) are 310 and 2510 respectively. (Arya and Bennet, 1976). Recently, it was shown that the annual rate of new gonorrhea infection in large African cities is 3,000 to 10,000 per 100,000 population or as many as one in every 10 people. By comparison, in the United State the annual incidence of gonorhea was 233 per 100,000 population in 1991 and in Sweden about 30 per 100,000 in 1987 (Aral et al., 1990, De schryver, 1990).

Several medications have been used before the era of antibiotic to treat gonorrhea e.g. application of salves and ointments and instillations of antiseptic into the urethra (Kampmerer, 1983). Standard recommendations have been issued by CDC for the treatment of gonorrhea and other sexually transmitted diseases and updated at regular intervals. These include penicillin G and probenecid, spectinomycin, ampicillin, amoxycillin, Tetracycline, Kanamycin, septrin, Cefriazone, cefuroxime, Quinoconoes e.g. ofloxacin ciprofloxacin, norfloxacin and combination therapy e.g. Ampicillin/Amoxycillin and Ampicillin/Sulbactam. However in many African countries, first choice antibiotics currently recommended by World Health Organisation (WHO) for gonococcal infections are neither available nor affordable to STD patients. Less expensive antibiotics show highly variable susceptibility and resistant patterns across the continent,

making them unsuitable for unlimited recommendation without regular surveillance (Francois, 1995).

World Health Organisation (WHO) estimates there are 183 to 327 million new cases of treated sexually transmitted diseases every year worldwide. Up to 94,000 of these cases occur in sub saharan Africa, which has the highest regional incidence of gonorrhea in the world.

Sexually transmitted diseases are receiving increased attention as a result of growing evidence of their association with the human immunodeficiency virus (Dallabetta, 1994). Recent evidence reveals that common sexually transmitted diseases (STDs) contribute to the spread of AIDs. Infectious such as chancroid, chlamydia, gonorrhea, syphilis, herpes genitalis makes a person more likely to become infected with HIV if exposed to AIDs causing virus (HIV) through sexual contact. These STDs may enhance HIV transmission because they increase the number of white blood cells which are both targets and sources of HIV in the genital tract and because genital inflammation may cause microscopic cuts that can allow HIV to enter the body. By altering hosts immune response, HIV may in turn change the epidemiology of STDs. Diseases causing vaginal and urethral inflammation are responsible for a larger

more often treated and cured (Cameron et al., 1989). STDs such as gonorrhea make it easier for HIV to pass from one person to another and may increase the risk of HIV transmission by two to nine times (Cleghorn et al., 1993; Kriess et al., Simonsen et al., 1988).

In view of the aforementioned observations on STDs and in particular gonorrhea and the possible link with AIDs, this work was designed to examine the prevalence of gonorrhea among patients attending A.B.U teaching Hospital STD Clinic complaining of urethritis, Cervicitis, dysuria with purulent discharges and to determine the incidence of HIV from confirmed cases of gonorrheal infections. These objectives will be achieved by:-

1. Screenng of symptomatic patients for Neisseria gonorrheae
2. Determininig the antibiotic susceptibility to commonly used antibiotics of the Neisseria gonorrheae isolates.
3. Screening the confirmed cases of gonorrhea for HIV.

The research will be a useful epidemiological information on the antibiotic susceptibility of gonococcal isolates, to serve as a guide to treatment and rational use of antibiotics owing to the fact that in Nigeria and most tropical countries adequate bacteriological service in most clinics and hospitals is lacking, prescriptions are mostly based on clinical judgements and these often are wrong. The findings should also contribute to the essential international reference data on the link between Human Immunodeficiency Virus (HIV) and

Sexually Transmitted Diseases especially gonorrhea which is a problem of worldwide concern.

CHAPTER TWO

LITERATURE REVIEW

Gonorrhea is a sexually transmitted disease caused by gram negative coci Neiseeria gonorrheae. At present, Neisseria species are classified in the family Neisseriaceae (Josephine, et al, 1992). Gonococcus are usually described as "kidney or bean" shaped, but may be oval or spherical as well, and may show arrangements other than classic diplococcal form. Using light microscopy, they are readily seen under oil immersion lens (Williams, 1994). Neisseria gonorrheae is a gram negative diplococcus that is oxidase positive. It affects both male and females. Neisseria gonorrheae is a capneophilic aerobe and does not survive for long outside the host because of its susceptibility to drying, heat and low concentrations of anions.

2.1 HISTORY OF GONORRHEA

Gonorrhea, a bacterial infection of male and female genital mucosa, has been endemic throughout the world for many centuries. Probably the earliest description of gonococcal urethritis can be found in the Bible in the Book of Leviticus.(William, 1981) The Egyptians were also familiar with the disease, but it remained for Galen (130AD) practicing in Rome during the golden age of Greek medicine, to give the disease its present name. He mistakenly believed that the discharge (rheos) was composed of semen (gonos). Thus gonorrhea means a flowing seed (Williams, 1981), but

Maimonides (1135 - 1204 AD) recognised that the discharge was not semen (Emil, 1990). Neisseria gonorrheae the causitive agent, was described by Albert Neisser in 1879 and first cultivated by Leistikow and Loeffler in 1882 (Emil, 1990; Holmes et al., 1984 and Knapp, 1984).

From the earliest history until modern days gonococcal urethritis has continued to be a problem of serious impact despite the advent of antimicrobial drugs specifically effective against the organism, it maintains an unenviable position as the most reportable infectious disease in the United States and the commonest sexually transmitted disease in tropical Africa (Osoba, 1981).

2.2 EPIDEMIOLOGY AND PREVALENCE OF GONORRHEA

An epidemic is an observed frequency of disease clearly in excess of normal expectancy. Sexually transmitted disease (STDs) particularly gonorrhea constitute a major medical and health problem worldwide as it presents with the following diseases or symptoms such as urethritis, epididymitis, proctitis, cervities, conjuctivities, prepubertal vaginitis, prostatitis, accessory gland infection, amniotic infection syndrome, chorioamnitis, premature rupture of membranes and premature delivery (William, 1984). Gonorrhea has been on the rise throughout the world since 1966. Significant increases have been reported to the world health organisation from Argentina, Austria, Bolivia, Canada, Chad, Columbia, Costa Rica, Denmark, Elsalvado, Ecuador, Finland, Gabon Germany,

HongKong, Israel, Iran, Iraq, Jamaica, Mali, Nigeria, Norway, Panama, the Philippines, Poland, Rwanda, Swaziland, Trinidad, and Tobago, the United States, United Kingdom and Venezuela. In developed countries e.g. the United States, despite the advent of antimicrobial drugs specifically effective against the organism, it maintains an unenviable position as the most reportable infectious diseases (Dallabeta and Hook, 1987). On the basis of under reporting and under diagnosis, the centre for disease control (CDC), reported that there were probably 2.6 million cases of gonorrhea in the United States during 1975, majority of which were in men. Two important factors responsible for high gonorrhea prevalence are the increasing resistance of Neisseria gonorrheae to available antimicrobial agents and a large reservoir of asymptomatic females and males who unknowingly transmit the disease to their sexual partners (Josephine, et al., 1992). It is commonly held that more men than women contract gonorrhea, but this may be an artifact of case finding and reporting. Willcox (1975) notes that the male female ratio of reported cases of gonorrhea is 3.1 or 4:1 in most developed countries. This is what one would expect if a relatively small group of highly promiscuous asymptomatic women were transmitting gonorrhea to a larger population of men who promptly become symptomatic and sought treatment.

Sexually transmitted diseases are a major health problem in both developed and developing countries, but prevalence rates apparently are far higher in developing countries, where STD

treatment is less accessible. Incidence rates of gonorrhea is higher in developing countries than in developed countries about 10 to 15 times (Wasserheit, 1989).

The review of work done in Africa indicates the high incidence of STDs despite the lack of reliable statistic on the matter. The prevalence figure available for rural and urban tropical areas are alarmingly high for gonorrhea and its complications (Osoba, 1981). In Uganda and Kenya prevalence rates of about 20% were noted among the females population. Prostitutes apparently cause few cases of gonorrhea in Europe and America but they are relatively more important in the overall epidemiology of gonorrhea in Africa and Asia (Willeox 1975).

In Nigeria, the prevalence of gonorrhea among asymptomatic women is about 50% and among prostitutes and female hospital patients is between 15% and 20% (Osoba, 1981). Also, Bello, et al., (1983) in a study conducted between 1977 and 1981 on 3089 patients, attenders of STD clinic in Zaria, reported post pubertal gonorrhea accounting for 28.1% i.e. 910 patients out of the total study population.

Youths have high rates of STDs. In study at Kenyatta National Hospital in Nairobi, for example, 23% of women ages 15 to 19 seeking antenatal care had gonorrhea chlamydia or herpes genitalis. The highest attack rate of gonorrhea in both men and women tend to get gonorrhea at an earlier age than men i.e. women ages 15 to 19 have the second highest

incidence of gonorrhea. The number of cases is rising most rapidly in teenagers (Reza, 1971).

In Africa there is a strong association between gonorrhea and reproductive failure in both sexes. For example in Teso district in Uganda, Arya and Taber (1973) found out that among men over 30 years about 44% of those with bilateral epididymitis were childless. Among women, salpingitis and pelvic inflammatory disease are the commonest complications of gonorrhea and frequent result in tubal occlusion and infertility.

.3 TRANSMISSIBILITY OF GONORRHEA

Gonorrhea is transmitted between individuals by direct, close usually sexual contact, but there have been reports of genital gonorrhea with out vaginal penetration as well as non-genital infections acquired by contamination with infected secretions (Frazier, et al 1979). People with large number of different sexual partners are at greater risk of contracting gonorrhea. Frequency of contact is also significant. Gonorrhea is transmitted 20 times more frequently in man with more than four sexual partners than it is among men with a single sexual partner (Holmes et al., 1970). Fifty to 90% of female sexual partners of infected men are infected after one exposure, 60% to 80% after four exposures (Holme et al., 1970; Hook et al, 1990). Gonorrhea was found in 70% of the frequent sexual partners but in only 26% of an infrequent or single sexual contacts of partners. Infection also occurred twice as

frequently among men having five or six exposures to an infected partner as among men having only one or two exposure (Brown, 1974). Gonorrhea can also be contracted on the toilet seat contaminated by an infected person. The incubation period for gonococcal urethritis after exposure varies from three to ten days (Williams, 1984). Mucosal infection usually causes inflammation with subsequent signs and symptoms that are useful in diagnosing gonococcal urethritis. Even though such inflammation usually subsides in two to four weeks without treatment, the host may remain carrier and potentially infectious (McCutchan, 1981). Extragenital gonorrhea also occurred without vaginal penetration (Frazier, 1979). These include oropharyngeal and anorectal gonorrhea and can occur as a result of rectal intercourse and fellatic, infections at these sites are more common in homosexual males and the patients may present with proctitis or acute pharyngitis and tonsillitis (Janda et al., 1980). There is also a risk of neonatal infection with Neisseria gonorrheae at the time of delivery in which the new born develops opthalmia neonatorum. In a study in Nigeria and Cameroon the risk of gonococal ocular infection in infants born to infected mothers was found to be 30 percent (Galega et al., 1984). Rarer neonatal consequences may be sepsis, meningitis or arthritis. Prevention of gonococcal opthalmia neonatorum is best attained by prenatal diagnosis and treatment of maternal infection along with treatment of her partner or by ocular prophylasis

by silver nitrate. The lower the overall transmissibility, the larger must be the number of contacts between infected and uninfected people to yield the observed incidence of gonorrhea.

2.4 COMPLICATIONS OF GONORRHEA

Gonococcal urethritis may spread by extension beyond the anterior urethra to cause significant local complications or it may disseminates widely via the lymphatics to produce distant disease. Thus, gonorrhea has many complications including epididymitis, urethral stricture, proctitis, pelvic inflammatory disease sterility and infertility.

2.4.1 EPIDIDYMITIS AND URETHRAL STRICTURE

The commonest complications of gonorrhea in men are urethral stricture and epididymitis. It is estimated that between 1% and 3% of men with five or more episodes of gonococcal infection develop bilateral epididymitis depending on the treatment given. In certain parts of Africa up to 80% of the practice of a urologist is taken up by urethral strictures and the management of reproductive failures resulting from STDs (Osoba, 1981). For example, Arya and Taber (1983) found out that among men over 30 years in Teso district Uganda about 44% of those with bilateral epididymitis were childless, a proportion significantly greater than 19% for those without epididymitis. Local extension to the posterior urethra, seminal vesides, or epididymis occurs frequently in men with untreated disease and may occur in even promptly

treated cases (Greenberg, 1979). Posterior urethritis in pre antibiotic days commonly led to urethral stricture and sterility, but this complication is rare in developed countries today (Engelbrecht et al., 1967). Epididymitis in younger men is frequently caused by gonococci but in men over 40 years, epididymitis genarally is caused by gram negative rods and may be associated with non-venereal urinary tract infections (Harrison, 1979).

2.4.2 DISSEMINATED GONOCOCCAL INFECTION (DGI)

Disseminated gonococcal infection is occasionally a concomitant of gonococcal urethritis, but more often it occurs in the absence of urethral symptoms (Holmes et al 1971; William, 1984). In small percentage of people with gonorrhea (0.5 to 3%), the organism spreads hematogenously to produce disseminated gonococcal infection (DGI). DGI is characterized by the presence of sparse rash on the extremeties and arthritis in one or more joints (Josephine et al., 1992; Holmes 1971).

At present gonococci is the major cause of septic arthritis in young adults. Although Neisseria gonorrheae may be isolated from the blood, skin lesions, or joint fluids of patients with DGI, often the organism is isolated only from genital, anorectal, or pharyngeal cultures of affected individuals with signs and symptom of DGI. DGI occurs more commonly in females than males, perhaps because there is a greater tendency for this organism to disseminate during menstruation and pregancy

(Morella, et al., 1976). In rare incidencies DGI may result in gonococcal endocarditis or meningitis. (Morello and Bonhoff, 1989; Josephine et al., 1992).

2.4.3 PELVIC INFLAMMATORY DISEASE (PID)

Neisseria gonorrheae are recovered from over three fourth of women with PID. In most women salpingitis occurs from a prior cervicitis that is most commonly attributable to Neisseria gonorrheae (Josephine et al 1992, Osoba, 1981). In some females with gonococal cervicitis, the infection ascends, resulting in salpingitis, pelvic peritonitis, or both complications refered to as pelvic inflammatory disease (PID) (Dallabetta 1987). Although other organisms may cause PID, gonococci are isolated from the cervices of 10 to 70% of affected women, which indicates that gonococci probably plays a major role at least in initial episodes of this disease (Dallabeta, 1987). The annual rate of salpingitis is 1.5% among women over the age of 25. It is estimated that 15 percent of women in the United States aged 30 years have had salpingitis (David, 1984). PID can cause infertility. Without treatment 55% to 85% of women with PID may become infertile. In some areas where PID is a commom cause of women with blocked fallopian tubes, 40 to 80% did not report that they had PID (Osoba 1981). In a Study of 86 patients with PID in Uganda, it was found that about one third harboured the gonococcus while 25% were infertile compared with 15% in a matched hospital control group. It was also noted that

pregnancy wastage, which occured in about 28.6% of all pregnancies was exceedingly high (Osoba, 1981). In tropical Africa with very high prevalence of gonorrhea, a large proportion of the female population will at one time or other have PID. In a study in Nairobi out patient clinic Kenya, 50% of patients with PID were found to have gonorrhea and about half of them were HIV seropositive (Dallabeta, 1987).

2.5 ANTIBIOTIC RESISTANT - GONOCOCCI

Resistance to penicillin in Neisseria gonorrheae was first recognised in the 1950s and was due to specific resistance genes (Lambert, 1992). In 1975 the first cases of beta-lactamase producing Neisseria gonorrheae were isolated in Phillipines followed soon thereafter by occurance in England and the United States. Penicillin resistant Neisseria gonorrheae have been causing disease in the United States for more than 30 years but the incidence of such strains has increased in certain groups. The number of reported cases in the United States jumped from 16,000 in 1986 to 25,000 in 1987. The ability to hydrolyze the B-lactam ring of penicillin like antibiotic is mediated by bacterial plasmid harbouring the gene for the enzyme B-lactamase, penicillinase producing Neisseria gonorrheae.

Since their initial description in 1976 (Phillips, 1976; Ashford et al, 1976), the prevalence of Neissseria gonorrheae capable of plasmid mediated B-Lactamase production (PPNG) has rapidly increased. In 1988, these strain composed

approximately 4.5% of all isolates of Neissrria gonorrheae in the United States, with more than 20% of the strains in Detroit, Miami and sections of New York city producing detectable levels of B-lactamase (Whittington and Knapp, 1988; 1990). While most strains of Neisseria gonorrheae in the United States do not as yet produce B-lactamase (PPNG) has rapidly increased . In 1988, these strains composed approximately 4.5% of all isolates of Neisseria gonorrheae in the United States, with more than 20% of the strains in Detroit, Miami and sections of New York city producing detectable levels of B-lactamase (Whittington and knapp, 1988; Morun and Zeilman, 1990). While most strains of Neisseria gonorrheae in the United States do not as yet produce B-lactamase, PPNG is a major public health issue in parts of Asia and sub-saharan Africa. In 1987, it was estimated that PPNG comprised 70 - 80% of goococceal isolates in Nigeria (Odugbemi, 1987), and in Manila, the Phillipines, 60% of the isolates of Neisseria gonorrheae examined during the period 1981 - 1985 produced detectable levels of B-lactamase (Antanio - velmonte et al., 1988).

Also, Osoba et al. (1981) reported sensitivity pattern of gonococcus to penicillin in Nigeria, the same study has been carried out in Uganda, Ethiopia, Kenya, South Africa and Swaziland. These show that over two thirds of the gonococcal strains in many tropical areas have diminished sensitivity to penicillin as a result of indiscriminate usage for prophylasis

and self medication.

It was found that relative resistance to antibiotics is already common in rural areas in which the majority of the African population live and that resistance to penicillin is even more common in rural areas than in cities. Evidence that this may result from the contraction of in adequate treatment especially in developing countries was obtained (Verhager et al., 1991; Melo et al., 1995). Such countries are being on immense reservoir of strains with a high level of resistance to penicillin.

2.6 TREATMENT OF GONORRHEA

Treatment of gonorrhea before the era of antibiotics include the application of ointments to the genitalia and the instillation of varius antiseptics solutions into the urethra. In 1936 to 1937 such remedies were discarded in favour of the newly discovered sulfa drugs. Over the next several years, however, resistance to sulfa drugs developed at an increasingly rapid rate. By 1943, the majority of isolates of gonococci were resistant to sulfadiazine (Kampmerer, 1983).

The discovery of penicillin began the current era of gonorrhea and in 1943 the first cases of gonococal urethritis were being treated successfully with penicillin. During the subsequent 40 years the amount of penicillin needed to eradicate the infecting gonococus has steadily increased. (Halverson et al., 1969). In 1972 the centres for Disease Control (CDC) issued a set of standard recommendations for the

treatment of gonorrhea and these include, 4.8 million units of aqueous procaine penicillin intramuscularly accompanied by 1gram orally of probenecid, spectinomycin 2gram injected intramuscularly, oral ampicillin 3.5 gram, amoxycillin 3 gram with 1 gram probenecid orally all as a single dose; tetracycline hydrocloride 500mg four times daily for one week. The World Health Organisation (WHO) global programme on STDs. in the population Reports (1993) requires any of the following for the treatment of gonorrhea,' cefurome 400mg orally; cefgtriazone 250mg intamuscula injection, ciprofloxacin 500mg daily; spectinpmycin 2grams all as a single dose, alternatively co-trimoxazole 480mg, 10 tables once daily for 3 days.

Quinolones have been extensively studied in the treatment of gonorrhea and most have been shoen to be effective (Hooper and wolfson, 1991) of loxacin is effect in gonowhoa including anorectal disease. Norfloxacin in an oral dose of 600mg and 600mg 4hour later seems to be effective. Using this regimen cider et al (1984) cured 100% of gonococcal urethritis amongst 59 US Navy men in the phillipines..Forty six were penicillin resistant but non-PPNG. A single dose of oral enoxacin 400mg is adequate treatment for rectal or pharynigeal gonorrhea. Ciprofloxacin (ciproxin) has been shown to be effective against gonococcal urethritis in a dose of 500mg bd for 7 days (Arya et al, 1986). and in a dose of 750mg b.d. for 4 days (Fong et al ., 1986). Pefloxacin (Abaktal) 400mg as a single

oral dose has been shown to be effective in the treatment of uncomplicated gonococcal infections in a clinical trial conducted in Ibadan by Bakare et al., (1996); of 64 cases 59 (92.2%) were PPNG and 5 (7.8%) were non-PPNG with cummulative percentage cure for all infections as 84.4%).

Also, the results of randomized study demonstrated that a single 1.5g intramuscular ceftriazone of uncomplicated gonococcal infections of genitourinary tract (Hellmann et al., 1995). Odugbemi et al., (1984) showed that clavulanic acid/amoxycillin and sulbactam - ampicillin combinations have invitroactivity against PPNG. Several earlier studies have indicated that the combination of ampicillin and sulbactam, wih or without probenecid, may be highly effective in treating both PPNG and non-PPNG strains (Kim et al. 1986; Odugbemi, 1987; Baddour et al., 1992). Clinical studies have confirmed this efficacy. Atia et al. (1983) used single dose sultamicillin 2g (plus probenecid) and cured 47% of men with uncomplicated gonococcal urethritis. Kim et al. (1984) treated 97 men with the same complaint (30% with PPNG) and achieved 96% cure rate using two doses of co-amoxiclar (Amoxycillin 3g/clavulanic acid 250mg) separated by 4 hour interval. cefotaxim, cetoxitim, cefaclor and sulfamethoxazole/trimethoprim are very efective against PPNG (Lancaster et al., 1981). A further group of compounds of increasing interest is the monobactams. Aztreonam is commercially available and it appears to be highly effective

against penicillin-sensitive and resistant gonorrhea in a single intramuscular dose of 1 gram (Miller et al., 1983; Evans et al., 1986). single dose of imipenem - cilistatin is also effective (Verdon et al., 1988).

2.7 ANTIBIOTIC SUSCEPTIBILITY TESTS

The principal method used by the laboratory to determine susceptibility of micro organisms to antimicrobial agents include the dilution test, agar plate dilution procedures and disk agar diffusion tests. The Kirby-Bauer disk diffusion method adopted officially in USA (Baver et al.1966, and used paper disk impregnated with antimicrobial agents. In the 1970's there was a general agreement on conditions of standardized antimicrobial susceptibility testing for common bacterial pathogens and that enabled the American College of pathologists to measure the accuracy of clinical laboratories in determining the susceptibility of bacteria by comparing their results with those obtained in reference laboratories (Fekete, 1993).

For invitro tests of susceptibility of Neisseria gonorrheae to antimicrobial agents, Putnam et al. (1992) described disk diffusion testing method and agar dilusion as the most commonly used methods in clinical laboratories. In disc diffusion methods three tests have been accepted as sufficiently reliable to give comparable results wwhen performed in different laboratories. They are : the method developed as a result of the International Collaborative Study

(ICS) sponsored by WHO , now practised mainly in Sweden (Stakes, 1987); the Kirby-Baver method adopted officially in the USA (Baver et al., 1966); and the comparative method practised in Britain and Other countries (Stokes 1987). Reproducibility of zone measurements was better with Kirby-Baver than with other methods but there were no significant differences in interpretation between the methods (Stokes and Ridgway, 1987).

2.8 PREVENTION OF GONORRHEA

Gonorrhea can be prevented by the regular use of condoms, spermicides, or prophylactic or post contact antibiotics, (Harison et al., 1979, cavalierid 'Ordo et al., 1994). Latex condoms are effective mechanical barriers used to prevent the passage of several infection agents including viruses, such as cytomegalvirus(CMV), Human Immunodificiency Viruses (HIV);; Chlamydia and bacteria such as Neisseria gonorrheae (Katzznelson et al., 1984; Judson et al., 1989). Females whose partners use condoms and male condom users themselves are protected against gonococcal infection (Barlow, 1977). Diaphragm, a barrier method of contracception, protects women against gonococeal infection. Rosenberg et al., (1992) found a 70% lower rate of infection for diaphragm users in comparison with no method of contraception and a 50% lower risk for diaphragm and sponge users in comparison with condom users. A protective effect of the diaphragm was also reported in a study conducted on prostitutes in Singapore (Bradbeer et

al. 1988). Diaphragm therefore, appears to have a protective effect against gonorrhea and non gonococal urethritis and cervical infections similar to that of condoms.

There was a consistent protective effect of spermicides on gonococcal infection. Spermicides are usually enclosed in media to show the progression of sperms and occlude the external uterine ostium. (Rosenberg and Gullub, 1992). Spermicides are not a single class of chemicals. For instance, nonoxyno (- (N-9) is classified as a surfactant, while benzalkonium chloride is a disinfectant and phenylmercuric acetate is an organic mercurial compound. In vitrobenzalkonium chloride has a direct inhibitory effect on HIV and N-9 inhibits the growth of several infective agents, such as Neisseria gonorrheae, Treponema pallidun and chlamydia trachomatis (Judson, 1989).

Another important measure that can be taken to reduce the occurance of gonorrhea is having a single faithful sexual partner, monogamy, or at least limitation of sexual contacts and exposures (William, 1984).

2.9 HUMAN IMMUNODEFICIENCY VIRUS (HIV) AND AIDS

In 1986, a sub committee of the international committee of taxonomy of viruses at the international congress on AIDs recommended that the sub-family of viruses that caused AIDs be named Human Immunodeficiency Virus (HIV) (Coffin et al., 1986). Because of the similarities in the clinical, biological and molecular properties of the HIV to lentiviruses, HIV was placed in the genus lentivirus sub-family lentiviridae of the family Retroviridae. Clinically, lentiviruses are usually associated with suppression of the immune systems. Presently, there are two types of HIV designated HIV-1 and HIV-2 - HIV-1 was isolated in 1983 and is common in the western World particularly USA and Europe. The existence of HIV-2 has been largely confirmed to west Africa and was isolated in 1986 from West African AIDs patients and from seropositive asymptomatic individuals (Clavel et al., 1987). More recently, HIV-2 has been found increasingly in Europe and Brazil and several cases mostly in immigrant West Africans, have been reported in the United States (Ayanian et al., 1989; Cortes et al., 1989). In comparison with HIV-1, there is lower viral burden and decreased CD4 cell killing with HIV-2. There is also good clinical evidence that rates of progression to AIDs and death are slower with HIV-2 than with HIV-1 (O'shanghnessy, 1994).

HIV infection can lead to variety of disease states, including an acute mononucleasis .like syndrome, prolonged asymptomatic infection, AIDs related complex, and frank AIDs

(Jaffe et al., 1983). according to the centers for Disease control (CDC) bulletin, 1982, AIDs is the occurance of a disease that is prodictive of a defect in cell mediated immunity, accuring in a person with no known cause for diminished resistance to that disease. Persistent antibodios to HIV usually develop within 1 week to 3 months of infection, but there have been report of delayed sero conversion of up to 3 years before antibodios to the virus appears (Imagawa et al., 1989). progression from HIV infection to AIDs differs among population groups and has charged over time. From several studies from united states it appears that in the absence of treatment , 40 to 50% of infection individual will progress to develop AIDs 7 to 10 years after infection (Moss and Bacchetti, 1989) and the majority of HIV infected individuals remain asymptomatic for at least 2 to 5 years.

After infection with HIV the immune defence mechanisms of the body is weakened and the body easily succumbs to various infections especially the opportunistic agents such as the viruses e.g cytomegalovirases, Herpes simplex virus and Epstein, Barr vius, bacteria e.g mycobacterium tuberculosis and Nocardia Species; fungi e.g candida species and Histoplasma Capsulatum;; protozoa e.g Pneumocystis carini, Toxoplasma gondi and cryptosporidium species (Gottlies 1983, Humburg, 1994).

A wide spectrum of clinical expressions could be produced as a result of HIV infection and the subsequent invasion of

the body by opportunistic infections which include fever lympha denopathy, macular rash, myalgia, arthralgia cough, diarrhea, fits, fatique, weight losss and neurological manifestatations (Osmond et al; 1987; Moss and Bachetti, 1989).

AIDs patients suffer from caposis sarcoma, pneumocystis carinii pneumonia, chronic diarrhea often caused by cryptosporidia, cryptococal meningitis, toxoplasmosis, encephalopathies and dementia as.well as anal or rectal carcinomas and B - lymphocytic lymphomas. In Africa, HIV infection and AIDs are also characterized by chronic diarrhea, a waste syndrome, resistant tuberculosis aggressive kaposis sarcoma and persistent herpes zoster (Quinn et al; 1986). Most AIDs patients in the united states have died from Pneumocystis carinii pneumonia, where as in Africa they frequently succumb to cryptococal meningitis or chronic dehydration secondary to severe diarrhea, a condition called slim disease (schnittman, 1989). AIDs is therefore, a complex of disease resulting from primary immune disorders induced by the causative agent, HIV.

2.9.1 GEOGRAPHIC DISTRIBUTION

The World Health Organization (WHO) has estimated that between 500,000 and 800,000 persons in the united states were infected with HIV as of 1988 (Osmond and Moss, 1989); Worldwide 5 million to 10 million persons by the year 1992. The infection in the first decade resulted in about 12 million infections. Twice as many will probably occur in its second

decade, with a total of at least 40 million people infected by the year 2000 (Phyllida, 1992). Early in 1993, the WHO estimated that around 13 million HIV infections had occured in adults since the begining of this scourge and at least one million children have been HIV infected around the world. An estimated 2.5 million AIDs cases have been reported by WHO by June 1993 in respect of different continents as given in the following estimates) sub-saharan Africa 1,775,000; Latin America and the carribean, 240,000 (9-6% western europe, 120,000 (4.8%); Eastern Asia, and central Asia, 3000 (0.12%), South and south east Asia, 30,000 (1.2%) and Australasia 21,000 (0.84%).

2.9.2: HIV PREVALENCE IN NIGERIA

AIDs was first diagnosed in Nigeria in 1986. Prior to isolation of HIV 2 in 1986, Nigeria was thought to be one of AIDs free zones of the world. The number of reported AIDs cases rose sharply from two cases in 1989 to 1,148 in 1994 (Nnorom, 1995).

But with the discovery of HIV -2, which has relatively longer incubation period it was clear that Nigerians are equally at risk of infection. Even at 1988, the WHO global AIDs incidence Inventory Reports, showed that cases were much less in Nigeria, at least comparatively. However, conservative estimates in the early 1990's indicate that over 100,000 Nigerians will develop full blown AIDS between 1991 to 1995, because over 0.5 million people were thought to be harbouring

the virus. On the occasion of the World AIDS day, December 1, 1993 coordinator of the National AIDs Control Programme, Dr. Abiola Tilley gyado, revealed that between 500,000 to 600,000 seropusitive cases have been reported in Nigeria based on random screening across the country (Meseda, 1993).

In 1991/92 the federal ministry of health and social services through Nigeria AIDs/HIV/STD control programme conducted a nation wide HIV sentinel survey . The result showed that the overall prevalence rate among the commercial sex workers (CSWs) out of 1,339 tested 234 were HIV positive (17.5%) where as out of 4,517 ante natal clinic attenders (ANC) screened 61 (1.4%) was recorded as positive. Usually the ANC is taken to represent the general population as they are not associated with any special risk factors that increase the transmission of HIV. The HIV prevalence rate for the general population in 1991/92 was 1.2% This meant that with about 600,000, Nigerians were infected with HIV. commercial sex workers are regarded as a high risk group as evidenced by a high prevalence rate (17.5%) (Tilley-gyado et al., 1995 Nnorom, 1995).

In 1993/94 the Federal Ministry of Health repeated the survey. This time the HIV prevalence rate increased from 17.5% to 21.3%) (a 22% increase) among the commercial sex workers where as the rate for ANC and hence for general population rose to 3.8% from 1.4% (a 217% increase). This means that by 1994 about 1.7 million Nigerians were already infected with

HIV. Also, the prevalence rate among STD patients is increaseing rapidly as from 4.6% in 1991/92 sentinel survey to 8.9% in 1993/94 (Nnorom,1995).

The prevalence rate of HIV was in decreasing order in Nigeria having commercial sex workers being greater than STDs patients followed by Tuberculosis patients and antenatal clinic attenders (CSW >STD > TB > ANC). The age specific prevalence rate ranges between 15 and 50 years. Females acquire the infection at a much earlier age 15 to 19 years as compared to males 20 to 29 years (Tilley - Gyado et al. 1995).

2.9.3. EPIDEMIOLOGY, PREVENTION AND CONTROL OF HIV INFECTION

HIV is transmitted through intimate sexual contact (homosexual, bisexual or heterosexual), infected blood and blood products (e.g through blood transfusion or through usage of infected needles, knife and skin piercing instruments), as well as from an infected mother to the unborn child either inutero or during passage of the baby through the birth canal. The HIV trasmission in Nigeria and other African countries is mainly by heterosexual contact.

A major route of sexual trasmission of HIV is anual intercourse. The anal- receptive partner is more susceptible to infection than is the insertive partner. The virus may infect the mucosal epithelial cells of the blood directly (Levy et al; 1989; Nelson et al, 1988) or gain access to other susceptible cells via injured areas in the mucosal living of the bowel. Transmission of the virus also occurs by vaginal

intercourse with the female showing the highest sensitivity Sexual Transmission of either kind is enhanced in the presence of other sexually transmitted diseases including gonorrhea, syphilis, and ulcerating diseases such as genital herpes and chancroid, as observed in African infected population (Susan and levy, 1992).

The major groups infected with HIV in the early stages of its spread in the united states and western Europe were homosexual (and bisexual) men, Haitian immigrants and intravenous drug users. The number of heterosexuals infected by sexual contacts with these groups is increasing, as is the number of babies born to HIV infected mothers (Levy, 1985), Quin et al; 1986). The other groups at risk of HIV infection have been haemophiliacs and blood transfusion recipients. But these have been possible (Levy et al., 1985; susan and Levy, 1992). There is no evidence that HIV can be trasmitted through insect bites, sharing of house hold utensils ordinary body contacts such as hugging and touching.

After the virus has gained entry into the human body, an initial incubation period of two to ten years persists during which the virus multiplies. The target cell for the HIV is the T4 Helper lymphocytes. The largerhans cells in the epidermis and the macrophage microcyte cells of the brains are also affected (Meseda, 1993, susan and Levy, 1992).

Presently, there is no vaccine to protect against HIV infection and there are no curative therapies. The best means

to prevent the spread of infection is through education.. Avoiding contaminated blood and blood products and intimate sexual contact with HIV infected persons condoms should be used during sexual activities involving potentially infected individuals and remaining faithful with a single non -infected sexual partners. Intravenous drug users must avoid sharing contaminated needles and syringes. Blood and blood products should be screened to eliminate infected individuals susan and Levy, 1992,).Certain drugs have been used for the treatment of HIV or to combat rapid turn over of the virus, these are called antiretroviral drugs consisting of nucleoside analogues, proteinase inhibitors, and reverse transcriptase inhibitors and many new compounds under development (Lipsky, 1996).

The first drug to be approved for use in HIV infection was zidovudine, an inhibitor of reverse transcriptase developed over 30 years ago. (Havlir and Richman, 1996) and is licenced by United States food and drug administration and recognised worldwide. This analogue of a standard deoxynucleoside is taken up by the cell and phosphorylated to a triphosphate (nucleotide) analogue, the active form of the drug (Zidovudine). Nucleoside analogues are known as chain terminators, since the DNA chain is terminated with incoporation of the dideoxynucleotide Termination of DNA synthesis prevents the virus from replicating and therefore causes viral stasis. Since the development of zidovudine, four

other drugs in this class have been approved for human use - zalcitabine (ddC), didanosine (ddL) Stavudine (D4T), and lamivudine (3TC) (Lipsky, 1996).

Inhibition of the virally produced proteanse known as "HIV proteinase" provides another therapeutic category in which a drug has been approved for treatment of HIV infection. Two of the protein products of HIV are precursors that are cleaved to mature protein products. This cleavage is catalysed by HIV proteinase. The released proteins are crucial for viral replication and include the protease itself as well as reverse transcriptase integrase, and structural proteins. If these proteins are not cleaved by the precursors proteins, then HIV is non-infectious (Morrow et al., 1994). The first inhibitor of HIV proteinase approved for clinical treatment was saquinavir, followed shortly by ritonavir and saquinavir. These drugs decrease viral load in patients (Bartlett, 1996; Kitchen et al., 1995 and Danner et al., 1995 and was approved by United States food and Drug Administration.

Reverse transcriptase can be inhibited by agents that are not nucloeside analoguis. Nevirapine, a dipyridodiazepinone, and tetrahydrobenzodiazepine (TIBO) derivatives bind to hydrophobic pocket in reverse transcriptase when this enzyme is complexed to DNA (Spencer et al., 1995). Nevirapine in combination therapy improves surrogate markers of HIV infection and has been recommended for clinical use (D'Aquila, et al., 1996).

Preliminary studies with another agent, atevirdine, showed improvement in CD4 counts when patients were treated with this agent and zidovudine (Reichman et al., 1995). Another promising antiviral approach is identication of agents that specifically kill HIV infected cells. A good example of this treatment is compound Q (trichosan thin), a subtance derived from the root of a chinese cucumber which has been shown to selectively kill HIV infected T-cells and macrophages invitro (McGrath et al., 1989).

There are many new antiviral agents for HIV infection under development. These include Zinc finger proteins which contains zinc, have finger-like loops, and bind to DNA and RNA. Benzamide compounds have beeen developed that seem to inhibit HIV virus through an interaction with these protains (Rice et al., 1995). Recently, endogenous substances from lymphocytes, chemokines, have been isolated which may interfere with a co-receptor involved for HIV infection (Bair et al., 1995; Balter, 1996). These substances may have therapeutic potential in combating infection with HIV. By then 10 million with HIV infection will have developed AIDS and 90% of them will be living in developing countries.

2.9.4. THE LINK BETWEEN STDs AND AIDS

By 1992 between 9 and 11 million adults and about one million children had been infected with the AIDs causing Human immunodeficiency virus (HIV), according to the World Health

Organization (WHO) estimates. Two thirds of these people live in developing countries. By the year 2000, 30 to 40 million will be infected (Phyllida, 1992). Sexually transmitted diseases are every where, Gonorrhea, syphilis and AIDs are the most widely known, but there are 20 other STDs. On average, estimated 685,000 people are infected every day with an STD (Khanna et al., 1991). WHO estimated there are 153 to 327 million new cases of treatable sexually transmitted diseases (STDs) every year world wide, at least one million of which will be HIV infection. Up to 94,000 of these cases occur in sub-saharan Africa, which has the highest regional incidence of gonorrhea in the world. (Wassaheit, 1992, Khanna et al., 1992). A critical health problem in their own right, STDs are receiving increased attention as a result of growing evidence of their association with the HIV.

The possible interactions between HIV infection and STDs include the following: 1. STDs may increase a persons ability to transmit or acquire HIV infection; 2). HIV may alter the clinical presentation, natural history, diagnosis and responsiveness to theraphy of STDs; 3). It is possible that STDs may influence the natural history of HIV infection (Dallabetta, 1994). A number of studies suggest that all of these interactions occur between HIV and some STDs like gonorrhea. Example in a study in Cotonou, Benin Republic (Alary et al., 1995) is the association between symptomatic gonococeal and chlamydia trachomatis infections and HIV

seropositivity in which there is up to 53.3% HIV seroprevalence. Also, gonococcal cervicitis, bacterial vaginosis, vaginal candidiasis, chlamydia trachmatis infection were found to be associated with HIV in study conducted in Atrica Viz; Kenya, Senegal, Zimbabwe and Uganda (Maton et al., 1995 Kiriat et al 1995, Mastad et al, 1995 and Jacinta, 1995). Studies in 1986 - 1989 among family planning and antenatal women in Nigeria showed STD prevalences to be: gonorrhea 5.2%, Chlamydia 6.5 - 11.0% and Syphilis 2.1%. A 1994 population based urban study among sexually active women showed 18% were infected with at least one STD (ESU-Williams Eka et al., 1995). In another study carried out in Amsterdam, the Netherlands, Johan et al., (1995) found out the incidence of STDs e.g. gonorrhea, early syphilis, pelvic inflammatory disease, chlamydia trachomatis infection, trichomoniasis, genital herpes and warts were found to be strongly correlated with HIV among drug using prostitutes. In Mai, Thailand out of 144 female commercial sex workers (CSWs) tested and found to have cervical gonorrhea, chlamydia infection, syphilis, trichomonas vaginitis, candida vaginitis, bacterial vaginosis, 43% were found to be HIV positive, in this case the STDs may be co-factors of HIV transmission (Craig et al., 1995). STDs enhance HIV transmission because they increase the number of white blood cells which are both targets and source of HIV in the genital tract because genital inflammation may cause microscopic cuts that can allow HIV to enter the body.

Diseases causing vaginal and urethral inflammation like gonorrhea are responsible for a larger share of HIV transmission (Wasserheit, 1992).

HIV infection in Nigeria is sexually transmitted infection with the sexually active sectors constituting the major pools of the infection. In this context, the demonstrated high prevalence among the commercial sex workers, STDs must not be myopically seen as a problem affecting only that group, but recognised along side all these factors that promote unprotected, casual and multiple sexual activities.

Also, people with HIV and STDs like gonorrhea and chancroid were found to fail treatment at least six times more often than in patients with STD alone and no HIV (Cameron, 1989). Findings from a Tanzanian study suggest that treating people for sexually transmitted diseases can dramatically reduce their risk of HIV infection. The number of new HIV infections was reduced by 40 percent among people who received STD treatment through a trial conducted by Tanzanians and British researchers in Tanzanian Mwanza region over the study period of two years, 11.6% of every 1,000 adults in the treated group of 6,000 adults became infected with AIDS virus as compared to 18.6% of every 1,000 adults in control group of 6,000. Thus, HIV enhances its own transmission with longer lasting STD symptoms, people with HIV infection are more likely to transmit HIV and increase the pace of AIDs epidemic (Wasserheit, 1992).

The review of workdone explained above showed clearly that the present position of gonococcal and HIV infections in tropical Africa and Nigeria in particular is very serious and the problems are enormous. There is therefore an urgent need of research to improve facilities for diagnosis and proper treatment of gonorrhea and enlightment campaign against HIV transmission.

CHAPTER THREE

MATERIALS AND METHODS

3.1.0 STUDY POPULATION

The study subjects were 200 sexually active males and females, 15-50 years of age, who reported at the Ahmadu Bello University Teaching Hospital Sexually Transmitted Diseases Clinic Zaria complaining of urethritis, or vaginal discharge between October 1995 and July 1996.

3.2.0 SAMPLES COLLECTION AND TREATMENT

Urethral and endo-cervical High vaginal swabs were collected from the patients with sterile cotton wool swabs. Smears were made on glass slides and gram-stained for gram-negative oval-bean shaped intracellular diplococci.

Oxoid blood agar base (Oxoid, limited.London) in dehydrated forms was prepared as directed by the manufacturer. Chocolate agar was formed by heating blood agar to lyse the red cells thus giving the brown media. Each swab collected was immediately innoculated on chocolate agar medium and incubated at 35-37^{0}c in an atmosphere containing 5-10% CO_2 (candle jar] for 24-72 hours. The growth of colonies 0.5-1mm in diameter which appeared grey to white, opaque, raised and glistening were identified as suspected Neisseria gonorrheae. The plates were kept for further tests which included antibiotic sensitivity, oxidase and carbohydrate utilization tests.. Neisseria gonorrheae in pure culture were grown on chocolate agar after 24 hours incubation, after which it was harvested

entirely from the surface of the plate into 0.3 ml of buffered salt solution (B.S.S) for carbohydrate utilization test.

Attempts were also made at isolating and identifying other microorganisms grown from the swabs.

3.3.0 ANTIBIOTIC SUSCEPTIBILITY TESTING

Tests of susceptibility of the Neisseria gonorrheae isolates to different commonly used antibiotics in gonorrheal treatment were carried out by standardized disk diffusion method. The following antibiotics, Penicillin, Tetracycline, Ampicillin, Gentamycin and Erythromycin (BDH chemicals Ltd., Poole, England) as multidisks, Cefuroxime and ceftriazone (Oxoid, Unipath Ltd., Basingstoke Hampshire, England) and Ofloxacin (Hoeschst Ltd., Marburg Germany) as single disks were used in the susceptibilty tests. The disks were stored at 80^0c in the refrigerator as instructed by the manufacturers.

The commercial antibiotics impregnated disks were placed on plates of sensitivity testing agar (chocolate agar) innoculated with the Neisseria gonorrheae isolates. After 24 hours incubation, the plates were examined for zones of inhibition. These were reported as sensitive (S) with inhibition zone radius greater than or equal to 3mm, intermediate (I) with zone radius less than 2mm (i.e, 3+, 2+, and 1+).

Control - Staphylococcus aureus (NCTC 6571) obtained from the Medical Microbiology Department, Ahmadu Bello University

Teaching Hospital were used as the control. The strains were maintained on nutrient agar slopes and stored at 4^0c from which sub-cultures were made for daily use.

3.4.0 COMFIRMATION OF ISOLATES

The gonococcal isolates were confirmed by oxidase test and carbohydrate utilization test using the criteria proposed by Brown (1974).

3.4.1 OXIDASE TEST

The oxidase reagent (Tetramethyl phenylenediamine dihydrochloride) manufactured by BDH Chemicals Ltd., Poole, England was measured in 0.1g amounts and dissolved in 10ml of distilled water and stored at 4^0c for use. A piece of filter paper was soaked with few drops of the oxidase reagent while a colony of the organism was then smeared on the filter paper. A change in colour to dark purple within 10 seconds of smearing the filter paper was indicative of presence of oxidase.

3.4.2 CARBOHYDRATE UTILIZATION TEST

Buffered Salt Solution (B.S.S) was prepared by suspending 0.04g of di-potassium hydrogen phosphate (K_2HPo_4), 0.01g of Potassium di-hydrogen phosphate (KH_2Po_4), 0.8g of potassium chloride (KCI) and 0.2ml of phenol red (1% aqueous) as an indicator in 100ml of distilled water. The solution was properly dissolved and stored at 2-8°c for use (Brown 1974). Twenty percent of stock solutions of various carbohydrates (Glucose, Maltose, Sucrose, and Lactose) manufactured by Oxoid

Ltd., London were prepared with distilled water. Two millilitres volume of each sugar was dispensed in small test tubes and stored at + 8^0C.

On drop of each carbohydrate solution to be tested and one drop of heavy bacterial suspension were added to tubes containing 0.1ml of buffered salt solution (BSS) and mixed. The tubes were incubated for 4 hours at 35^0C in water bath and observed for acidification. Development of visible yellow colour within 4 hours of incubation of an oxidase positive diplococci was recorded as a positive result confirmatory for Neisseria gonorrhoae. Tubes containing buffered salt solution (BSS) only and bacterial suspension served as control which were not expected to show any colour change.

3.5.0 HIV SCREENING

Five millilitres of blood were collected by veni puncture from patients with confirmed cases of gonococcal infections into sterile universal bottles and stored at 2-8^0c until they was needed for HIV testing. The serum was carried to HIV screening centre for antibody detection against HIV 1 and 2. Enzyme Linked Immunosorbent Assay(ELISA, Wellcozyme 1 and 2) reagents (Murex Diagnostic Ltd., England) were used for screening as instructed by the manufacturer.

Detection of antibodies to HIV by ELISA is done by the reacting the patient serum and the purified viral antigen (wellcozyme 1 and 2). An enzyme conjugated secondary antibody is added, and the presence of antibodies in the sample was

detected by a colour reaction after addition of enzyme substrate. The colour reaction was quantified by spectrophotometry using the Reader and the result appeared on the printer. The confirmatory test for the detection of the presence of HIV antibodies was performed on each positive sample by Double antibody ELISA. The results were recorded as reactive (Positive) or non-reactive (Negative) for HIV 1 and 2 antibodies.

CHAPTER FOUR

RESULTS

4.1 Study Group:

Out of the 200 symptomatic patients screened for gonococal infection, one hundred and fifty eight (79%) were males and fourty two (21%) were females. The range of their years were between 15 and 50 years. The age and sex distribution of the study subjects were shown in table I.

Table I: AGE AND SEX DISTRIBUTION OF THE STUDY SUBJECTS

			MALE		FEMALE	
AGE GROUPS (YRS)	TOTAL	% TOTAL	NO SAMPLE	%	NO SAMPL	%
15 - 20	34	17.0	22	14.0	12	28.60
21 - 25	57	28.5	39	24.70	18	42.85
26 - 30	61	30.5	54	34.20	7	16.70
31 - 35	27	13.5	25	15.80	2	4.80
36 - 40	16	18.0	14	8.90	2	4.80
41 - 45	2	1.0	1	0.65	1	2.40
46 - 50	3	1.5	3	1.90	0	0
GRAND TOTAL	200	100	158	100	42	100

Patients between the age of 26 - 30 (30.5%) has the higher percentage of attendance while less patients attended the clinic between 40 - 50 (2.5%) years of age. More males attended the clinic between age of 26 - 30 (34.2%) contrary to females which recorded more patients between the age of 21 - 25 (42.85%).

4.2 MICROBIOLOGICAL RESULTS

The prevalences and distribution of gonocuccal infections by age and sex among the study population is shown in Table II, More males (17) has positive gonococcal infection then eight (8) recorded in females.

TABLE II: PREVALENCE OF GONOCOCCAL INFECTION BY AGE AND SEX AMONG THE STUDY POPULATION

		MALE			FEMALE			
AGE GROUP (YRS)	TOTAL	No. SAMPLED	No. POSITIVE	% PREVALENCE	No. SAMPLED	No. POSITIVE	% PREVALENCE	TOTAL % PREVALENCE
15 - 20	34	22	0	0	12	4	25.0	12.50
21 - 25	57	39	4	10.25	18	2	11.10	10.70
26 - 30	61	54	7	12.96	7	2	28.60	20.80
31 - 35	27	25	6	11.10	2	0	0	5.55
36 - 40	16	14	0	0	2	0	0	0
41 - 45	2	1	0	0	1	0	0	0
46 - 50	3	3	0	0	0	0	0	0
GRAND TOTAL	200	158	17	35.00	42	8	65.00	49.5

Out of the 200 Urethral and high vaginal swab examined, oval been shaped gram positive diplococci were seen in 25 samples after Gram staining and all yielded a growth of grey, Opaque, raised and glistening colonies of Neisseria gonorrheae when cultured on diocolate agar. Neisseria gonorrheae was isolated in 12.5% (25) of the sample tested (Appendix 1) The isolates were also found to be oxidase positive and utilised glucose as shown in appendix 2.

The Prevalance and distribution of gonococcal infection by age and sex among the study population is shown in table 2. More males (17) has positive gonoccal infections as compared with eight (8) in females. The Neisseria gonorrheae shows the highest prevalence between the age of 26 - 30 years for males patients 7/17 and 4/8 between the age of 15 - 20 years among the females patient. No positive gonorrhea infection were found in both males and females above the age of 35 years. Females were found to harbour gonorrhea in the early age than males due to the early exposure especially in the Northern part of Nigeria. Females also has higher percentage prevalence (65.0%) or 8/42 than males (35.0%) or 17/158. Total % prevalance is highest between the age of 26 - 30 years (20.8%) in both sex.

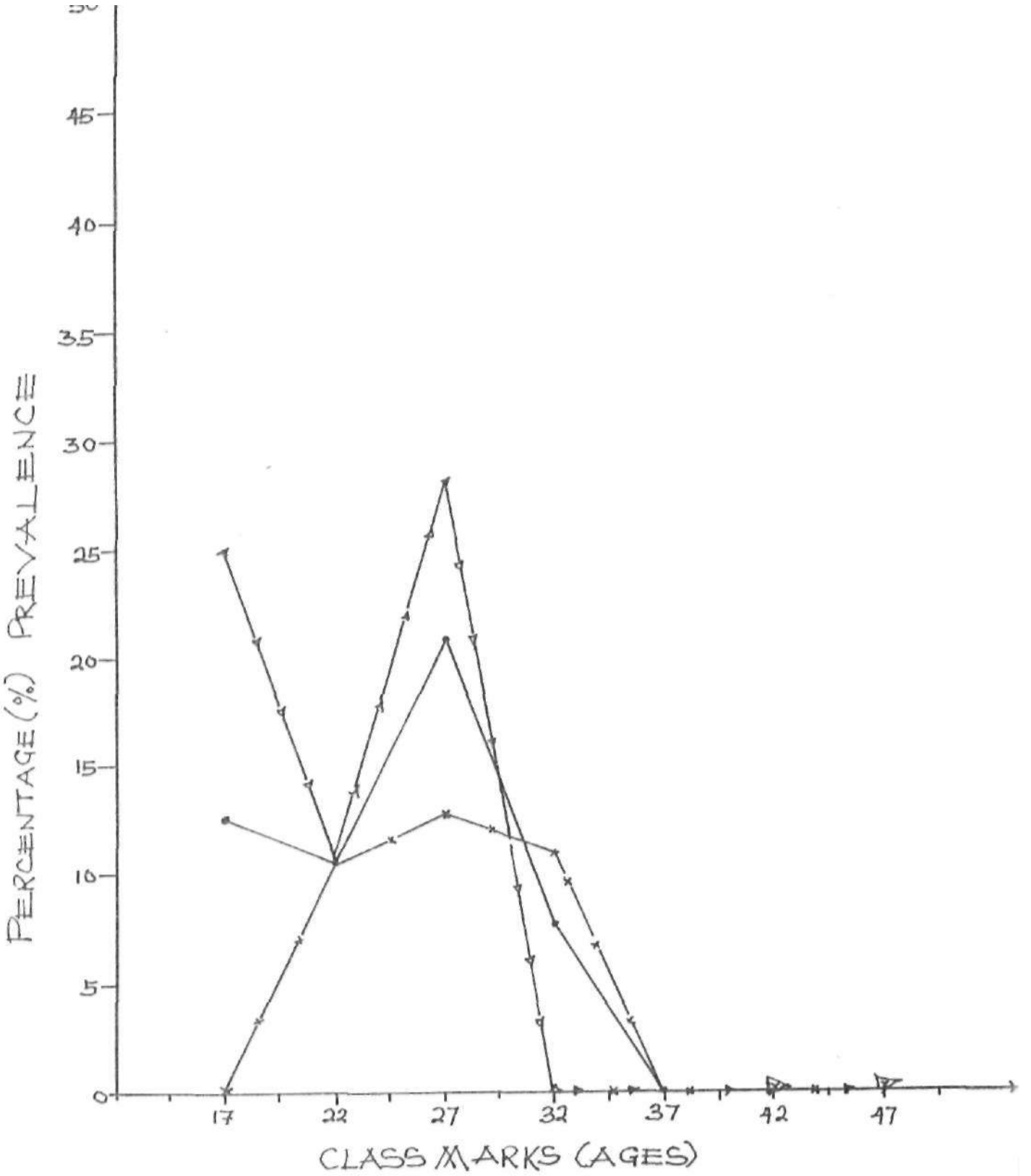


Fig 1: DISTRIBUTION OF CASES OF GONOCOCCAL INFECTIONS BY SEX AND AGE AMONG THE STUDY SUBJECT.

KEY: —×—×— MALE
—▷—▷— FEMALE
—•—•— TOTAL

Apart from Neisseria gonorrheae other STDs were also isolated and identifed. This included Staph aureus (25.5%) and candida albicans (5.5%) all these were identified by wet preparations, Grumstain and culture. Table 3 shows % prevalence of Neisseria gonorrheae infection in relation to infectious agents of other STDs

Table 3: PREVALENCE OF NEISSERIA GONORRHEAE INFECTIONS IN RELATION TO AGENTS OF OTHER STDs

PATHOGEN	TOTAL No. SAMPLED	No. POSITIVE	% PREVALENCE (N = 200)
Staph aurpus	200	45	22.5
N gonorrheae	200	25	12.5
C albicans	200	11	5.5

N/B No organism isolated in 119 specimens.

The total cases of staphylocuccus aureus infections out of 200 sample examined is 45, thus representing 22.5%, N gonorrheae 25 representing 12.5% and that of C albicans is 11 which is 5.5%. T hus the total number of observed STD was 81 (40.5%) including N. gonorrheae. No organism was isolated in 119 (59.5%) specimens:

The overall sensitivity of such antibacterial agent was penicillin 3(12%), Ampicillin 4(16%), Tetracyclinic 6 (64%), Erythromycin 17(68%), Gentamycin 18(72%), Cefuroxime 16(64%), Ceftriazone 24(92%) and Ofloxacin 25(100%). Sensitivity and resistance of N. gonorrheae isolates to the antbiotics tested is shown in table 4.

TABLE 4: SENSITIVITY AND RESISTANCE OF N. GONORRHEAE ISOLATES TO THE ANTIBIOTICS TESTED				
ANTIBIOTIC	NO SENSITIVE	% SENSITIVITY (N=25)	NO RESISTANT	% RESISTANCE (N=25)
PENICILIN	13	12	22	88
AMPICILLIN	4	16	21	84
TETRACYCLINE	16	64	9	36
ERYTHROMYCIN	17	68	8	32
GENTAMYCIN	18	72	7	28
CEFUROXIME	6	64	9	36
CEFTRIAZONE	24	92	2	8
OFLOXACIN	25	100	0	0

The most effective antibiotic against N. gonorrheae is ofloxicin with 100% sensitivity followed by ceftrizone, Gentamycin, Erythromycin, Tetracycline and Cefuroxime which has over 60% sensitivity. The least active of the agents were penicillin and Ampicillin with sensitivities of less than 20% and resistance of 88% and 84% respectively.

Quinoloness (Ofloxacin) and cephalosporins (ceftriazone and cefuroxime) were the most active agents, aminoglycoside (Gentamycin), Erythromycin and Tetracyclines were moderately active while penecillins (Penicillin G and Ampicillin) were poorly active against Neisseria gonorrheae isolates as found in this study. (Appendix 3). The staphylococcus aureus control strains showed 100% sensitivity to all the anitibiotic tested.

Out of the 25 positive cases of gonococcal infections 4(16%) were HIV seropositive, all of which were HIV.1 serotype. The incidence of HIV among Neisserias gonorrheae patients were shown in table 5.

TABLE 5: INCIDENCE OF HIV AMONG NEISSERIA GONORRHEAE PATIENTS

PATHOGEN	TOTAL No. SAMPLED	TOTAL No. POSITIVE	% POSITIVE
N. GONORRHEAE	200	25	12.5
HIV	25	4*	16.0

* ALL THE HIV POSITIVE CASES WERE HIV.1 SEROTYPE

HIV positive patients received more frequent antibiotic treatment due to HIV induced immuno suppresion compared to non HIV subjects this lead to resistance to commonly used antibiotics. In this study Neisseria gonorrheae isolates from the HIV positive subjects were found to be resistant to commonly used antibiotics (ampicillin and penicillin) and all were sensitive to the less common and more expensive ones e.g ceturoxine, ceftriazone and ofloxacin (appendix 3).

More men with gonococcal infections were found to be HIV sero positive as compared to their female counterparts. Out of 17 Neisseria gonorrheae cases in males 3(17.6%) were HIV seropositive while females has only 1(12.5%) incidence. Percentage privalenace by sex and age of HIV amongst Neisseria gonorrheae patients of the study population is shown in table 6.

TABLE 6: PREVALENCE BY SEX AND AGE OF HIV AMONGST PATIENTS WITH NEISSERIA GONORRHEAE OF THE STUDY POPULATION

		MALE			FEMALE			
AGE GROUP (YRS)	No. SAMPLED	No. +VE FOR N.GONORRHEA	No. +VE FOR HIV	% PREVILANCE	No. +VE FOR N.GONORRHEA	No. +VE FOR HIV	% PREVILANCE	TOTAL TO PREVILANCE
15 - 20	4	0	0	0	4	0	0	0
21 - 25	6	4	1	25	2	1	50	33
26 - 30	9	7	1	14.2	2	0	0	11
31 - 35	6	6	1	16.6	0	0	0	7
36 - 40	0	0	0	0	0	0	0	0
41 - 45	0	0	0	0	0	0	0	0
46 - 50	0	0	0	0	0	0	0	0
GRAND TOTAL	25	17	3	17.6	8	1	12.5	16

Men and women aged between 21 - 25 years have the highest % prevalance having one HIV seropositive patient each. There is one HIV seropositivity in men among 26 - 30 and 31 - 35 years of age. All the positive samples were confirmed by double antibody ELISA test.

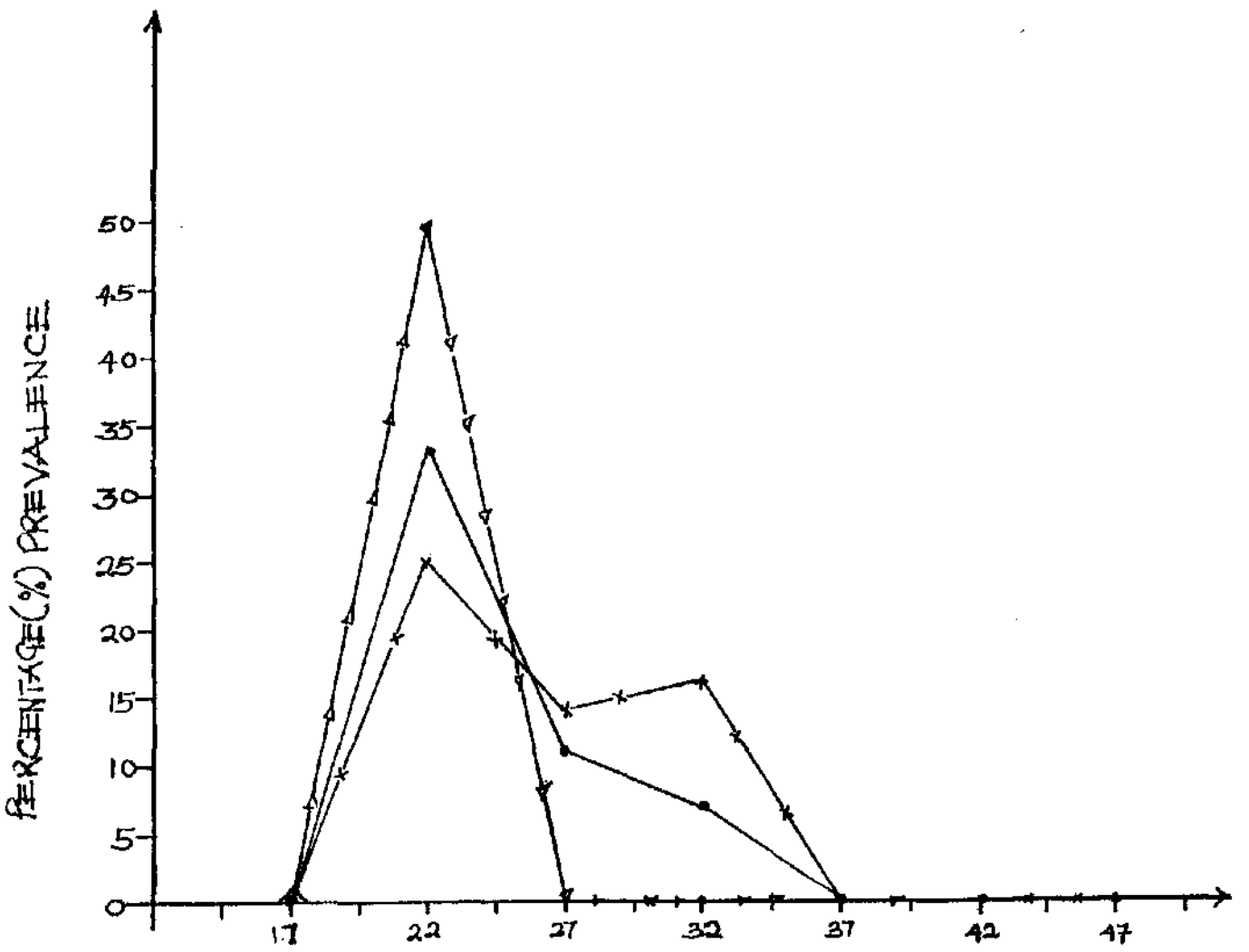


FIG2: THE DISTRIBUTION OF HIV POSITIVE CASES AMONGST GONORRHEA PATIENTS BY SEX AND AGE.

KEY:
——x——x—— MALE
——▸——▸—— FEMALE
——•——•—— TOTAL

CHAPTER 5

DISCUSSION AND CONCLUSION

The result of this study demonstrate that Neisseria gonorrheae and HIV are linked with each other. The incidence of HIV among gonorrheae patients was very high (16.0%). Apart from Neisseria gonorrheae among the patients who attended the clinic within the study period, evidence of infections with other STDs varying from syphilis, candidiasis, staphylococcal. infections and chlamydia trachomatis were found.

It is commonly held that more men than women contract gonorrhea from different findings. Willcox (1975) noted that the male to female rate of reported case of gonorrhea is 3:1 in most developed countries. This study is consistent with Willcox's findings as 17/25 of males had gonorrhea against 8/25 of the females. This is what one would expect if a relatively small group of highly promiscuous asymptomatic women were transmitting gonorrhea to a larger population of men who promptly became symptomatic and sought treatment. The asymptomatic infection with Neisseria gonorrheae known to occur in males, is generally considered to be a rare phenomenon (Handsfield et al., 1974).

In the present study, over 80% of the Neisseria gonorrheae isolates were resistant to penicillin and ampicillin. Previous studies (Osoba, et al., 1977; Montefiore et al, 1987 and Obaseiki Ebor et al., 1985) in Nigeria and Ethiopia showed comparable resistance rates of 64% to 87% for those agents.

These high resistance levels may reflect the epidemic proportions of this disease in some areas and also may be partly due to the large African rural population which may harbour a reservoir of resistant strains common to tropical countries (Osoba, et al., 1977). There may be a common basis of multiple drug resistance, perhaps due to alterations of the permeability of the outer envelop. The high rate of resistance of Neisseria gonorrheae to commonly used antibiotics like Penicillin and Ampicillin could be due to the indiscriminatory use of antibiotics, self medication and as prophylasis.

Other agents such as Tetracycline, Erythromycin, Gentamycin and Cefuroxime used in this study showed over 60% sensitivity to Neisseria gonorrheae isolates. Oral Cefuroxime (axetil) as lipophilic ester of cefuroxime was found to be highly effective against Neisseria gonorrheae (Reichman et al., 1985; Wanas and Williams, 1986) as found in this study. Quinolones e.g. Ofloxacin have been shown to be effective against gonorrhea. Ceftriazone have been recommended for treatment of gonorrhoea by CDC and WHO as a standard therapy against gonorrhea. In this study 100% of the isolates were found to be sensitive to ofloxacin and 92% to Ceftriazone. These results are in general agreement with the literature which has shown over 90% cure rate (Ledbedeff and Hockman 1980) of gonorrhea and anorectal diseases.

studies have demonstrated an association between HIV and most of the classic sexually transmitted diseases e.g gonorrhea, syphilis, genital warts and trichomoniasis. The most consistent association is between HIV-1 and gonorrhea, syphilis and chancroid (Dallabeta, 1994). This study agrees with Dallabeta's findings as the HIV-1 sero prevalence among gonorrhea patients is very high but non of the HIV positive patients carry HIV-2 senotype. All the Neisseria gonorrheae isolates in HIV positive patients were found to be resistant to commonly used antibiotic e.g. penicillin and ampicillin and sensitive to less common ones (i.e. Ceftriazone and ofloxacin). This is because HIV positive patients received more frequent antibiotic treatment than HIV negative ones.

In conclusion, this study has shown a correlation between the presence of Neisseria gonorrheae and HIV seroprevalence among patients attending STD clinic at A.B.U. Zaria. Antibiotics such as ofloxacin and ceftriazone appears to offer a viable alternative to older agents, such as penicillin and ampicillin in the treatment of gonococcal infections, this will therefore serve as guide to treatment and rational use of antibiotics to Neisseria gonorrheae infections.

The data presented here suggests that among the patients analyzed, the clinical diagnosis of Neisseria gonorrheae is independently associated with HIV seropositivity. The possibility that Neisseria- gonorrheae could act as co-factor for the heterosexual transmission of HIV has important

implication. Although other genital infections have been implicated as co-factors, none is as prevalent as gonorrhoea as found in this study. HIV and STDs are linked by their primary mode of transmission and the sexual behaviours that put people at risk of acquiring them, parallel prevention strategies such as condom promotion, and risk reduction education could reduce both STD and HIV. STD programmes also stress early diagnosis and effective therapy in individuals, lower prevalence of STD in communities and eventually reduced incidence of HIV infection. The special STD clinic like the one at Ahmadu Bello University Teaching Hospital Zaria contributes to the patients personal health and welfare and treat all STDs, also, it offers HIV testing and councilling, promotes health education and allows STDs research as like the one carried out here, these may be of help in further limiting the spread of STDs like gonorrhea and HIV.

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APPENIX 1: CULTURE RESULTS OF SAMPLE COLLECTED AND ANALYSED

TEST No.	SEX	AGE	ORGANISM ISOLATED
1	M	40	Candida Albicans
2	m	40	No growth
3	M	22	No growth
4.	M	28	No growth
5.	M	23	No growth
6.	M	32	No growth
7.	M	18	No growth
8.	F	21	C albicans
9.	F	24	No growth
10.	M	25	Staph aureus
11.	M	20	Staph aureus
12.	M	27	N gonorrheae
13.	M	27	Staph aureus
14.	M	25	No growth
15.	M	26	Staph aureus
16.	M	26	Proteus species
17.	M	30	Staph aureus
18.	M	30	No growth
19.	M	35	N gonorrheae
20.	M	27	No growth
21.	M	39	No growth
22.	M	34	No growth
23.	M	27	No growth
24.	F	20	N gonorrheae
25.	F	20	Staph aureus
26	M	21	Staph aureus
27.	M	21	No growth
28.	M	30	No growth

29	M	14	Staph aureus
30	F	25	No growth
31	F	25	Staph aureus
32	M	15	N gonorrheae
33.	M	25	N gonorrheae
34.	M	32	No growth
35.	M	31	Staph aureus
36.	M	17	No growth
37.	M	31	Staph aureus
38.	M	30	Staph aureus
39.	M	30	N gonorrheae
40.	M	27	No growth
41.	F	31	N gonorrheae
42.	F	30	N gonorrheae
43.	M	30	Staph aureus
44.	M	30	No growth
45.	M	30	Staph aureus
46.	M	20	No growth
47.	M	22	No growth
48.	M	24	No growth
49.	M	38	Candida albicans
50.	F	33	Candida albicans
51.	F	28	No growth
52.	M	26	Streptococcus species
53.	M	26	Staph aureus
54.	M	35	No growth
55	M	32	Staph aureus
56.	M	40	No growth
57.	M	22	No growth

58	M	25	No growth
59	M	35	No growth
60			
61	M	35	N gonorrheae
62	M	35	Staph aureus
63.	M	27	Staph aureus
64.	M	27	N gonorrheae
65	M	26	No growth
66.	M	25	No growth
67.	M	26	Staph aureus
68.	M	29	Staph aureus
69.	M	40	Candida species
70.	M	25	No growth
71.	M	36	Staph aureus
72.	F	22	No growth
73.	M	35	N gonorrheae
74.	M	15	No growth
75.	M	27	Staph aureus
76.	F	22	N gonorrheae
77.	M	20	Staph aureus
78.	M	26	N gonorrheae
79.	M	19	No growth
80.	M	29	No growth
81.	M	29	No growth
82.	M	26	Staph aureus
83.	M	45	Staph aureus
84	M	25	No growth
85.	M	40	No growth
86.	F	20	Staph aureus

87	M	25	No growth
88	M	28	No growth
89	M	42	No growth
90.	M	35	N gonorrheae
62	M	35	Staph aureus
63.	M	27	Staph aureus
64.	M	27	N gonorrheae
65	M	26	No growth
66.	M	25	No growth
67.	M	26	Staph aureus
68.	M	29	Staph aureus
69.	M	40	Candida species
70.	M	25	No growth
71.	M	36	Staph aureus
72.	F	22	No growth
73.	M	35	N gonorrheae
74.	M	15	No growth
75.	M	27	Staph aureus
76.	F	22	N gonorrheae
77.	M	20	Staph aureus
78.	M	26	N gonorrheae
79.	M	19	No growth
80.	M	29	No growth
81.	M	29	No growth
82.	M	26	Staph aureus
83.	M	45	Staph aureus
84	M	25	No growth
85.	M	40	No growth
86.	F	20	Staph aureus

87	M	25	No growth
88	M	28	No growth
89	M	42	No growth
90.	M	28	No growth
91	M	32	No growth
92.	M	26	Staph aureus
93.	M	26	No growth
94	M	22	No growth
95.	M	29	No growth
96.	M	30	Staph aureus
97.	M	22	No growth
98.	M	26	Klebsiella spp
99.	F	36	No growth
100	M	22	No growth
101	M	20	Staph aureus
102	M	35	No growth
103	M	25	N gonorrheae
104	M	28	No growth
105	M	39	No growth
106	M	22	N gonorrheae
107	F	25	Normal vaginal flora
108	F	20	Candida albicans
109	M	28	No growth
110	M	29	No growth
111	M	35	No growth
112	M	18	No growth
113	M	26	No growth
114	M	24	Staph aureus
115	M	20	No growth

116	M	28	No growth
117	M	35	No growth
118	M	26	No growth
119	F	40	Staph aureus
120	F	24	Staph aureus
121	F	24	No growth
122	F	26	No growth
123	M	23	No growth
124	F	17	Staph aureus
125	F	21	No growth
126	M	25	No growth
127	M	27	No growth
128	M	24	No growth
129	M	23	No growth
130	M	30	Staph aureus
131	M	35	N gonorrheae
132	M	35	Staph aureus
133	M	35	N gonorrheae
134	M	29	No growth
135	M	25	No growth
136	F	20	N gonorrheae
137	M	22	No growth
138	M	29	No growth
139	M	26	No growth
140	M	35	No growth
141	M	26	No growth
142	M	35	No growth
143	M	30	No growth
144	F	21	Candida albicans

145	F	20	N gonorrheae
146	M	28	No growth
147	M	40	No growth
148	M	20	No growth
149	M	24	Staph aureus
150	M	27	N gonorrheae
151	M	27	Staph aureus
152	M	35	No growth
153	M	35	Hemolytic streptococcus
154	F	18	No growth
155	M	20	No growth
156	M	25	No growth
157	M	25	Staph aureus
158	M	23	N gonorrheae
159	F	22	Staph aureus
160	F	22	Candida albicans
161	M	26	N gonorrheae
162	M	45	No growth
163	M	27	Staph aureus
164	M	27	No growth
165	M	25	N gonorrheae
166	M	34	No growth
167	F	27	No growth
168	M	20	No growth
169	M	18	No growth
170	M	19	Staph aureus
171	M	32	No growth
172	M	20	No growth
173	M	18	No growth

174	M	21	No growth
175	M	51	No growth
176	F	26	Candida albicans
177	F	25	Candida albicans
178	M	25	No growth
179	M	30	No growth
180	F	25	No growth
181	F	20	No growth
182	M	15	No growth
183	F	27	No growth
184	F	21	No growth
185	M	21	No growth
186	M	36	No growth
187	M	50	No growth
188	M	50	No growth
189	F	20	Candida albicans
190	F	17	Staph aureus
191	F	27	N gonorrheae
192	M	24	No growth
193	M	24	No growth
194	M	24	No growth
195	M	33	Staph aureus
196	F	21	No growth
197	M	24	Staph aureus
198	M	19	No growth
199	M	18	No growth
200	F	23	No growth

APPENDIX II: BIOCHEMICAL CHARACTERISTIC OF N GONORRHEAE ISOLATES

TEST NO	CARBOHYDRATE UTILIZATION TEST				ORGANISM IDENTIFIED
	GLUCOSE	MALTOSE	LACTOSE	SUCROSE	
12	+ve	-ve	-ve	-ve	N. gonorrheae
19	+ve	-ve	-ve	-ve	N. gonorrheae
24	+ve	-ve	-ve	-ve	N. gonorrheae
32	+ve	-ve	-ve	-ve	N. gonorrheae
33	+ve	-ve	-ve	-ve	N. gonorrheae
39	+ve	-ve	-ve	-ve	N. gonorrheae
41	+ve	-ve	-ve	-ve	N. gonorrheae
42	+ve	-ve	-ve	-ve	N. gonorrheae
61	+ve	-ve	-ve	-ve	N. gonorrheae
64	+ve	-ve	-ve	-ve	N. gonorrheae
73	+ve	-ve	-ve	-ve	N. gonorrheae
76	+ve	-ve	-ve	-ve	N. gonorrheae
78	+ve	-ve	-ve	-ve	N. gonorrheae
103	+ve	-ve	-ve	-ve	N. gonorrheae
106	+ve	-ve	-ve	-ve	N. gonorrheae
118	+ve	-ve	-ve	-ve	N. gonorrheae
131	+ve	-ve	-ve	-ve	N. gonorrheae
133	+ve	-ve	-ve	-ve	N. gonorrheae
136	+ve	-ve	-ve	-ve	N. gonorrheae
145	+ve	-ve	-ve	-ve	N. gonorrheae
150	+ve	-ve	-ve	-ve	N. gonorrheae
158	+ve	-ve	-ve	-ve	N. gonorrheae
161	+ve	-ve	-ve	-ve	N. gonorrheae
165	+ve	-ve	-ve	-ve	N. gonorrheae
191	+ve	-ve	-ve	-ve	N. gonorrheae

APPENDIX III - SENSITIVITY AND RESISTANCE OF NEISSERIA GONORRHEAE ISOLATES TO THE ANTIBIOTIC TESTED AND HIV SEROSTATUS

			ANTIBIOTIC SUSCEPTIBILITY									
T/N	SEX	AGE	ISOLATE IDEN.	PEN	AMP	TCN	GENT	ERYT	CXM	CRO	OFX	HIV SEROSTATUS
12	M	27	N GONORRHEAE	R	S	S	S	S	R	S	S	-VE
19	M	35	N GONORRHEAE	R	R	R	S	S	S	S	S	-VE
24	F	20	N GONORRHEAE	R	R	S	S	S	S	S	S	-VE
32	F	15	N GONORRHEAE	R	R	S	S	S	R	S	S	-VE
33	M	25	N GONORRHEAE	R	R	RS	R	R	S	S	S	+VE
39	M	30	N GONORRHEAE	R	R	R	S	S	S	S	S	-VE
41	F	35	N GONORRHEAE	R	R	S	S	S	S	S	S	-VE
42	F	30	N GONORRHEAE	R	R	R	S	S	R	S	S	-VE
61	M	35	N GONORRHEAE	S	R	S	S	S	S	S	S	-VE
64	M	27	N GONORRHEAE	R	R	S	R	R	R	S	S	-VE
73	M	35	N GONORRHEAE	R	R	S	R	R	S	S	S	-VE
76	F	22	N GONORRHEAE	R	R	S	S	S	S	S	S	+VE
78	M	26	N GONORRHEAE	R	R	R	R	R	R	S	S	+VE
103	M	25	N GONORRHEAE	R	R	R	S	S	R	S	S	-VE
106	M	22	N GONORRHEAE	R	R	S	S	S	S	S	S	-VE
117	M	35	N GONORRHEAE	R	R	R	R	R	R	S	S	+VE
131	M	35	N GONORRHEAE	R	R	R	S	S	S	S	S	-VE
133	M	35	N GONORRHEAE	R	R	S	R	R	S	S	S	-VE
136	F	20	N GONORRHEAE	R	R	S	R	R	S	S	S	-VE
145	F	20	N GONORRHEAE	R	R	S	R	R	R	S	S	-VE
150	M	27	N GONORRHEAE	R	R	S	R	R	R	R	S	-VE
158	M	23	N GONORRHEAE	S	S	S	S	S	S	S	S	-VE
161	M	26	N GONORRHEAE	S	S	R	S	S	S	S	S	-VE
165	M	25	N GONORRHEAE	R	R	S	S	S	S	S	S	-VE
191	F	27	N GONORRHEAE	R	R	R	S	S	S	S	S	-VE

PEN - PENICILLING, AMP - AMPICILLIN, TCN - TETRACYLINE, GENT - GENTAMYCINE

ERYTHR - ERYTHROMYCIN CXM - CECUROXIME CRO - CETTRIAZONE, OFX - OFLOXACIN,

S = SENSITIVE, R = RESISTANT.