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Genital Parasitic Infections and their Role in HIV Transmission

Ozoko Tochukwu Chinedu

Department of Medical Microbiology and Parasitology, Delta State University, Abraka, Nigeria.

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Genital parasitic infections include those which are sexually transmitted primarily and those while may not be contracted via sex but may develop a genital syndrome in their course. These genital syndromes cause inflammation and ulceration and by these increase the risk of HIV transmission. More so their epidermiological factors also favour the transmission of the virus. The genital infections discussed are Trichomniasis, Pediculosis pubis, Scabies, Genital Schistosomiasis, Genital amoebiasis, Bancroftian filariasis, and cutaneous Leishmaniasis. An integrated approach is therefore necessary to control both the menace of these infections and the spread of HIV and is here advocated.

Key words: Genital Parastitic Infections, HIV Transimission, Nigerian population.

Parasitic infections have a worldwide distribution but are more common in the tropics and the third world.(Sweet L. R and Gibbs R .S, 2002)

Reproductive tract infections cause a Lot of suffering from Infertility, Still Birth, Brain Diseases, Blindness, General Ill Health Leading to Social, Economic And Spiritual Loss.

The recent demonstration that treatment of Sexually Transmitted Diseases(STDs) reduced the transmission of HIV has re-emphasised the importance of these diseases.(Eddleston M, Pierini S, 1999)

The HIV Pandemic

With its attendant immunosuppresive effects tend to alter the course and pathology of many diseases including parasitic diseases.(Harms and Feldhmeier, 2002)

On the other hand these parasitic diseases affecting the genital tract also affect HIV transmission.

Some genital parasitic infections are sexually transmitted, others are not but may be transferred via sex in certain conditions.

The AIM of this discussion

Is To Outline The Pathology Inflicted On The Genital Tract By These Parasites And The Role Of These Parasitic Genital Syndromes In HIV Transmission.

Known sexually transmitted parasitic infections include

Trichomoniasis

Pediculosis Pubis

Scabies

Trypanosomiasis Can Also Be Transmitted Sexually Though There Is Little Information On The Occurrence Of Genital Pathology In Man

Giardiasis Can Be Transmitted Via Anal Sex

* To whom all correspondence should be addressed.

Parasitic diseases with genital manifestations include

Genital Schistosomiasis
Genital Amoebiasis
Filariasis
Cutaneous Leishmaniasis (Richens J, 2004)

Pathologic and clinical effects of these parasites on the genital tract

Trichomoniasis

Trichomonas vaginalis is a sexually transmitted pea shaped anaerobic protozoan parasite infecting about 3 million new persons each year. Its prevalence correlates with overall level of sexual activity of the specific group under study

It is reported to affect5% of women attending Family Planning Clinics
13-25% Of Women Attending Gynaecological Clinics
50-75% Of Prostitutes
7-56% Of Women Attending Std Clinics

Note that up to half of infected women and most infected men are asymptomatic

The organism adheres to the genital epithelium where it becomes flattened and causes superficial lesions without invasion of host tissues. It causes low grade inflammation of the vaginal walls and punctate haemorrhagic lesions in the exocervix

Its presence is associated with the loss of vaginal lactobacilli and increase ph(5.5-6.0), Normal Is (3.8-4.4)

Clinically, The features in women—vaginal discharge,
Vulvovaginal Irritation
Dyspareunia
Dysuria
In Men———Urethritis

Mainstay of diagnosis is either by the microscopic examination of wet mounts or The culture of vaginal, or urethral or prostatic secretions or semen.

Treatment is with the 5-Nitroimidazoles .(Sweet L .R and Gibbs S .R, 2002)

Scabies

This highly contagious infestation with the Ítch Mite' *Sarcoptes Scabei Var Hominis* is transmitted by person to person contact including sexual contact.

The characteristic itch and Papulonodular skin lesions are due to sensitization reaction to mite excreta deposited by the female *Sarcoptes* in the burrows it has made beneath the stratum corneum of the epidermis.

These lesions as well as genital ulcers which they occasion often appear in the Scrotum And Penis of infected males.

Other parts of the body where the lesion may appear include—Axilla, Under The Breasts, Volar Aspects Of The Wrists, Groin, and Between Finger Spaces.

Immunity of the host and associated scratching tend to limit infestation to less than 15 mites per person.

However in cases of debilitating Illness involving Immune suppresion like Glucocorticoid use, Neurologic/Psychiatric illnesses and HIV, there may be hyperinfestation with thousands of mites– a condition known as Crusted Scabies or Norwegian Scabies. Treatment is with benzyl benzoate. (Eddleston M and Pierini S, 1999)

There may be secondary infection with Nephritogenic Streptococci.

Pediculosis pubis

The causative agent *Phthirus Pubis* is transmitted sexually and close Body Contact.

As many as 33% of infested individuals may habour other STDs

The organism resides primarily in pubic hair, It Pierces The skin to take a blood meal, injecting saliva and defecating at the same time.

It causes marked pruritus of all affected areas and erythematous macules, papules and excoriations. There may be secondary staphylococcal infections . Treatment is with 0.5% malathion liquid. (Eddleston M and Pierini P,1999)

Filariasis

Two species of filarial worm cause genital disease in humans. Much the most important is *Wuchereria bancroftii*, which accounts for 90% of filarial infections and is estimated to infect 100 million people in the tropics.(Richens J, 2004)

Of these, 40% have disfiguring manifestations and 27 million men are estimated to suffer from genital deformity.Genital morbidity in women is much rarer. (Dreyer G .B, 1998)(Bernard P *et al.,* 2000)

Bancroftian (lymphatic) filariasis has been ranked the second leading cause of disability in the world by the World Health Organization

(WHO) and ranked second to HIV in Haiti as a public health issue for the community. (Eberhard *et al.,* 1996).

Onchocerciasis, caused by *Onchocerca volvulus*, is remembered by most students of tropical medicine for an unusual complication known as the "hanging groin," caused by a combination of inguinal adenopathy and skin atrophy that results in hanging folds of skin containing lymph nodes. Minor deformities of scrotal skin may also develop (Gyapong *et al.,* 1994).

Recent surveys in endemic areas reported hanging groin in 14% in Nigeria and pendular scrotum in 19% in Ethiopia.(Nmorsi *et al.,* 2002, Mengistu *et al.,* 1996)

Filariasis occurs in Africa, Asia, South America, the Caribbean, and the Pacific. Transmission is through mosquitoes that transmit larvae that develop into adult worms in the human host.

Recent studies have shown it often possible to identify nests of adults worms by ultrasound. The worms display a characteristic movement termed the "filarial dance" (Amaral *et al.,* 1994).

In men the lymphatics of the spermatic cord are a favoured location.

The adult worms release microfilaria into the blood in large numbers in the early part of the night, thus making detection of microfilaria in a night blood sample a convenient tool for diagnosis.

The presence of infection leads initially to asymptomatic lymphangectasia (Dreyer *et al.,* 2000). The death of adult worms provokes acute inflammation and lymphatic dysfunction and the late effects of disease result from superimposed bacterial infection in areas of lymphatic dysfunction.

The specific clinical features that result from these processes in the genital area are described in the next page.

The diagnosis is usually made by demonstrating the presence of microfilaria in peripheral night blood samples, for which a variety of techniques are available (Simonsen, 2003). Adult worms within the scrotum can be demonstrated by ultrasound with a 7.5 MHz transducer (Amaral *et al.,* 1994). Demonstrating microfilaria becomes more difficult in late stage disease. Antibody and antigen detection techniques are available in special centres. One study conducted in an endemic area showed that 37% of men initially reported as amicrofilaraemic in a 60 µl capillary sample could be shown to have filariasis by testing larger blood volumes or by scrotal ultrasound for adult worms (Dreyer *et al.,* 1996).

Diethylcarbamazine (DEC) in three divided doses of 6 mg/kg/day for 12 days kills adults and microfilariae. The death of worms can provoke quite intense systemic and local reactions but does not generally require withdrawal of treatment. DEC treatment alone does not produce regression of hydroceles (Bernard *et al.,* 2000). Ivermectin is effective only against microfilariae and has an important role in control programmes. Surgery for scrotal elephantiasis produces much more satisfactory results than surgery for elephantiasis of the legs (Ollapallil JJDA, 1995). Studies from Ghana have shown that surgery for hydrocele brings marked improvements in physical and social wellbeing, increases capacity for work and community participation, and merits much greater attention than it has so far received in endemic areas. Aspiration followed by sclerotherapy with tetracycline offers a useful alternative method for dealing with thin walled hydroceles (Musa, *et al.,* 1995). Good skin care and prompt treatment of bacterial skin infections is important to stop the disease progressing.

Male genital manifestations of infection with *Wuchereria bancrofti*

Lymphangiectasia of lymphatics round the spermatic cords

Earliest sign of infection. Detectable by ultrasound in 80% of men found to have microfilaraemia.

Acute hydrocele

Develops when adult worms die naturally or as a result of therapy.

Chronic hydrocele

Detectable in up to 40% of males in areas hyperendemic for filariasis.

Chylocele

Collection of chyle that forms when a lymphatic ruptures into a hydrocele.

Lymph scrotum

Superficial scrotal lymphangiomatosis which may ooze chyle through deformed scrotal skin.

Acute inflammation of scrotum and penis

May be triggered by death of adult worms or superimposed bacterial infection. Tender scrotal nodules or irregularity of spermatic cords may be felt.

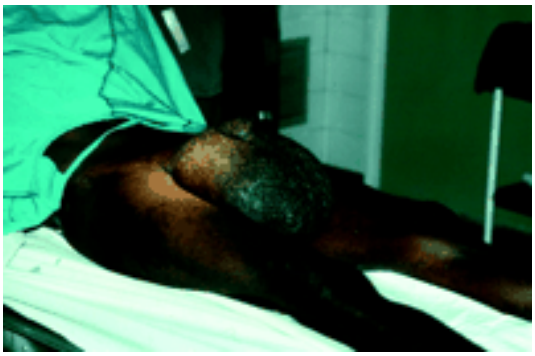
Elephantiasis of scrotum

Late hypertrophy and fibrosis that results from repeated bacterial infections. Urine flow not affected.

Inguinal adenitis

Develops acutely when adult worms die. Also triggered by bacterial infections in genitalia or legs. Occasionally filarial abscess develops.

Scrotal bancroftian filariasis in a patient in Papua New Guinea (Richens J, 2004)



Schistosomiasis

Schistosomiasis comprises a group of helminth infections characterised principally by extensive egg shedding into the bladder (mainly *Schistosoma haematobium*) or rectum (mainly *S mansoni* and *S japonicum*) by adults worms residing in nearby venous plexuses.

The inflammatory reaction to soluble egg antigens released through pores in the walls of eggs that become lodged in tissue produces a wide array of symptoms, notably haematuria and bloody diarrhoea, and sequelae such as periportal fibrosis.

Genital complications of schistosomiasis are less well known but may have important implications for control of cervical cancer and HIV in women. (Poggensee G and Feldmeier H, 2001)(Feldmeier H *et al.,* 1994)

Both sexes may develop genital complications but, in contrast with filariasis, the genital morbidity is much greater in women than men.

Schistosomiasis is acquired by exposure to water colonised by various species of snail which act as intermediate hosts and which release into the water motile miracidia capable of penetrating human skin.

The disease is believed to infect 193 million people in the tropics, with 85% of infections occurring in Africa. Each of the *Schistosoma* species that infects humans has a specific distribution related to local snail ecology.

Schistosomiasis can manifest a as genital tract disease both in males and females.

Female Genital Schistosomiasis and its Clinical Features(Poggensee G and Feldmeier H, 2001)

Site and clinical manifestations

*Fallopian tubes——Infection can simulate pelvic inflammatory disease and lead to infertility and ectopic pregnancy

*Uterus———Disturbed menstruation, fetal loss

*Placenta——Second trimester abortion

*Cervix————Ulceration, growths, sandy patches, cervicitis, discharge, post-coital bleeding, dyspareunia

*Vagina——Growths, ulcers, sandy patches, recto-vaginal and vesico-vaginal fistulae

*Vulva———Swelling, ulceration, wart-like growths, pruritus, clitoral hypertrophy

S. haematobium is the predominant worm but *S. mansoni* is also capable of migrating sufficiently to produce genital lesions. Feldmeier *et al.,* have estimated that 6–27% of females with intestinal schistosomiasis may also develop genital lesions (Poggensee *et al.,* 2000); (Feldmeier *et al.,* 1998).

The extent of genital morbidity arising from *S. japonicum* infection appears to be much less (Qunhua *et al.,* 2000). Confusion of cervical lesions with carcinoma and of vulval lesions with genital warts may occur. “Sandy patches” of the vagina and cervix which result from subepithelial calcification of ova are especially distinctive lesions (Helling-Giese *et al.,* 1996)

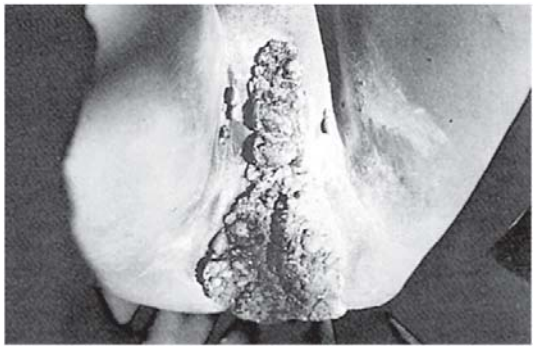
It has been postulated that FGS exacerbates the effects of human papillomavirus infection, leading to swifter development and spread of cervical cancer. Many women in Africa develop cervical cancer under the age of 30 and

Tanzanian studies have indicated high rates of FGS in the youngest women.

Women with FGS may develop multiple mucosal lesions and this, coupled with the intensity of inflammation associated with schistosomiasis, could make FGS an even more potent co-factor for HIV transmission than sexually transmitted infections, which often cause less mucosal disruption and inflammation. (Harouny and Pedersen, 1998; Ville *et al.,* 1991)

Genital schistosomiasis

(Goldsmith *et al.,* 1993; Gilles *et al.,* 1987; Wyatt, 1998).



Diagnosis of FGS

The diagnosis is best made by the quantitative compressed biopsy technique (QCBT) which gives a better yield than histology or examination of urine specimens.

A wet smear technique offers a means of rapid diagnosis in some cases.

Eosinophil cationic protein (ECP) is elevated in patients with FGS and testing for the presence of ECP is being explored as a possible method of screening (Poggensee *et al.,* 2001; Swart and Van der Merwe, 1987; Poggensee *et al.,* 1996).

Male genital schistosomiasis

The ability of schistosomiasis to cause bleeding and egg deposition within semen was first clearly demonstrated by the intrepid Claude Barlow (Barlow and Meleney, 1949) who deliberately infected himself with cercariae in 1944 and then observed the development of haemospermia and the appearance of schistosome ova in his own semen.

MacKenna *et al.,* reported how seven men from New Zealand attended a sexual health clinic in (Mckenna *et al.,* 1997) Christchurch complaining of either yellow discoloration , reduction in semen volume, or consistency . All the men had swum in Lake Malawi while travelling in Africa. Treatment with single dose of praziquantel led to recovery in all cases.

Other reports have described the development of haemospermia and lumpy semen in men with schistosomiasis.

Calcifications of the seminal vesicles and prostate may be observed on ultrasound.

There is report of a rapid diagnosis of schistosomiasis in a young adult male who attended a sexual health clinic in the U.K complaining of lumpy semen after returning from work in Africa where he reported swimming in Lake Malawi (Richen, 2004; Feldmeier *et al.,* 1999, Lewis *et al.,* 1996;Corachen *et al.,* 1994)

He produced a fresh specimen of semen in the clinic which confirmed his description. One of the lumpy areas was squashed under a cover slip and eggs containing wriggling larvae were shown to the patient before starting therapy.

There are a small number of case reports of lesions of schistosomiasis developing within the testes (simulating carcinoma or causing infarction), epididymis, and the penis. Male infertility resulting from such lesions however are rare. (Vilan *et al.,* 1997; Githae, 1992; Steinberger *et al.,* 1975; Kazzaz and Salmo, 1974; Badejo *et al.,* 1978; Girgis and Nassef, 1980).

Genital amoebiasis

The classic effects of infection with *Entamoeba histolytica* are the development of a colitis that gives rise to amoebic dysentery, colitis, and liver abscess when organisms enter the portal system. *E histolytica* has also been reported as a cause of genital ulcer.

In a review of 148 case reports of genital amoebiasis confirmed by observation of *E histoloytica* published between 1924 and 1997, Antony and Lopez-Po noted that 85% of reports were of genital infection in females, including infants (Antony and Lopez-po, 1999).

Female genital amoebiasis is characterised by a foul, bloody vaginal discharge. Cases of salpingitis caused by this organism has been reported (Calore *et al.,* 2002). In one third abdominal pain was reported and 8.1% had genital ulceration, often mimicking carcinoma of cervix,

although in some cases amoebiasis and carcinoma have been found together.

Involvement of uterus and tubes has also been reported; 92% of cases were diagnosed in cervical cytology specimens and the remainder by ulcer biopsy (Mhlanga *et al.,* 1992; Othman and Ismail, 1993; Calore *et al.,* 2002).

Eighty six per cent of cases in men presented as a painful, discharging ulcer , again often mimicking carcinoma and the remainder with discharge or dysuria.

The diagnosis in men was made by biopsy, culture, smear, or wet preparation. In addition to these methods serological tests and nucleic acid amplication tests are now available to diagnose amoebiasis.

Genital amoebiasis lesions generally respond swiftly to a standard course of metronidazole treatment (800 mg three times daily for 5 days).

Neglected cases have progressed to necrotising vulvitis requiring radical vulvectomy.

The infection can be sexually transmitted and sexual partners of patients with genital amoebiasis should be examined and offered treatment (Citronberg and Semel, 1995; Mylius and Tenseldam, 1962).

Genital amoebiasis in a patient from papaua new guinea

Cutaneous leishmaniasis affecting the genitals

The leishmaniases are a group of diseases caused by protozoa of the *Leishmania* genus that affect 1–2 million people per year between latitudes 45° north and 32° south (Richen, 2004).

Each species has an animal reservoir and can be transmitted to humans by sandfly bites. Following inoculation organisms are taken up by macrophages where they are able to resist degradation. The ensuing release of cytokines and cell mediated immune response to infection leads to the development of lesions.

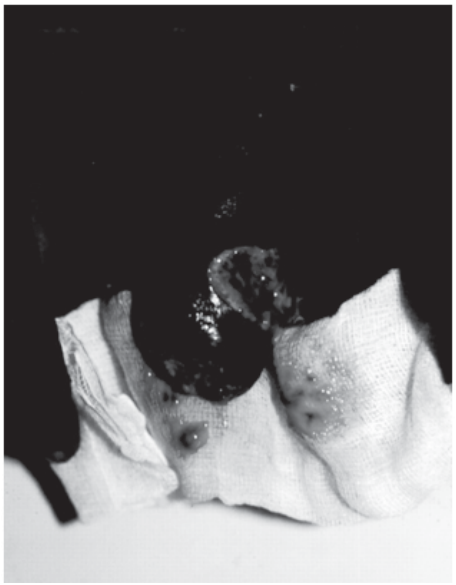
Cutaneous leishmaniasis is usually characterised by chronic localised ulceration developing at the site of inoculation. Genital lesions are rare but have been described in South America among miners and farmers. One vulval lesion has been reported (Cabello *et al.,* 2002; Castro-coto *et al.,* 1987; Blickstein *et al.,* 1993).

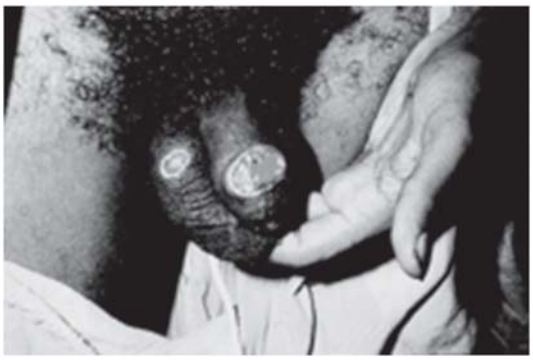
The diagnosis is confirmed by demonstrating the presence of *Leishmania* amastigotes in smear or biopsy material. Culture and polymerase chain reaction methods can increase detection rates.

Treatment should be undertaken with expert advice and will usually be either systemic or local pentavalent antimonial therapy depending on the the species and clinical picture. Mild cases caused by certain species such as *L peruviana* may resolve spontaneously.

Cutaneous leishmaniasis of penis and (Richen J, 2004)

Role of genital parasitic infection on transmission of hiv infection





Leishmaniasis and HIV transmission

As Seen Above The Genital Lesions Of Cutaneous Leishmaniasis Can Act as aPortal of Entry of the Virus

Th1-Cell Competence Is Defective In HIV Infection And Provides Afavourable Environment For The Manifestation Of Primary Visceral leishmaniasis Or For The Reactivation Of Latent Leishmanial Infection Acquired Many Years Earlier. (Harms and Feldmeier, 2002)

Schistosomiasis and HIV infection

In Genital Schistosomiasis, egg-induced ulcerative lesions can be the portal of entry for the virus. Infact there is recent pathophysiological, immunological and epidemiological evidence suggesting that genital schistosomiasis, is a risk factor for the transmission of HIV and presumably also alters the natural history of HIV infection in a deleterious way (Feldmeier *et al.,*. 1994, 1995; Poggensee *et al.,*. 1999, 2000).

Genital schistosomiasis in males also points at an increased risk of HIV transmission. In male adolescents and adults in Madagascar S. haematobium caused inflammation of the prostate and of the seminal vesicles in most patients (Leutscher *et al.,*. 2000).

As infection with S. haematobium in males induces a chronic inflammation in the pelvic genitals, it can be hypothesized that, in analogy to the situation in bacterial urethritis, there will be increased viral shedding in the semen in HIV co-infected individuals.

Filariasis and HIV infection

Gopinath *et al.,* (2000) observed that the replicative capacity of HIV is significantly enhanced in peripheral blood mononuclear cells from patients with untreated lymphatic filariasis.

Consequently, untreated patients with lymphatic Filariasis and co-infected with HIV could be at risk for rapid progression to AIDS once infected with HIV.

Moreso with rapid replication and the attendant epididymo-orchitis in filariasis, there will be higher concentration of the virus in semen facilitating transmission

STDs and HIV Infection

HIV Itself Is An STD, So Epidermiological Factors Which Affect The Transmission Of STDs Also Affect That Of HIV

Several studies suggest that treating reproductive tract infection may help prevent the transmission of HIV.

Two ways proven to reduce HIV transmission from adult to adult are syndromic treatment of STDS (Tanzania) and government promotion of condom use (Uganda and Thailand)

Parasitic diseases of the genital tract represent not only a nidus for transmisive entry but also may portend a dissemination of the parasite outside commonly encountered compartments

Points of note concerning HIV transmission

The virus appears to concentrate in the seminal fluid especially in conditions where there is increased lymphocytes and monocytes in the fluid as in genital inflammatory states like urethritis and epididymitis

The virus appears also in cervical and vaginal fluid. A history of STDs is strongly associated with HIV transmission and in this regard there is close association between genital ulceration and transmission with respect to both susceptibility to infection and infectivity. Furthermore non ulcerative inflammatory STDs like Trichomoniasis, show a close association with increased risk of transmission of the virus.

Importance of an intergrated approach to management

It is essential that control tools against parasitic diseases complement other tools currently being used against the pandemic. Efforts to control these infections and limit their capability in HIV transmission should include: (Harms and Feldmeier, 2002; Eddleston and Pierini, 1999)

1). Correct diagnosis
2). Effective early treatment
3). Education on avoidance of contact and prevention of transmission
4). Promotion and provision of condoms
5). Tracing, treating and counselling of sexual partners
6). Appriopriate clinical follow up.

It is essential that patients be treated when first seen and counselled to modify their sexual behaviour

Summary

In Summary These Parsitic Genital Tract Infections Cause The Std Symptoms Viz

Urethral discharge
Genital ulcers

Inguinal bubo
Scrotal swelling
Vaginal discharge
Lower abdominal pain

The Lesion Causing Most Of These Symptoms Increase The Risk Of Susceptibility To , And Infectivity With The Hiv Virus

A Multidisciplinary Approach Involving Physicians, Parasitologists And Venerologists Is Again Emphasised

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