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Genitourinary Complications of Schistosomiasis

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Abstract

Background*: Urinary schistosomiasis caused by S. haematobium is a parasitic disease endemic in many parts of tropical Africa, Middle East and South-West Asia and is associated with significant morbidity and mortality. The genitourinary complications of the disease are organ-specific and result from the immune response of the body to the chronic presence of the trapped eggs in the host tissues. The objective of this review was to elucidate the pathogenesis of the complications of schistosomiasis affecting the urogenital organs and the common treatment modalities.*

Methods: *Electronic literature search on PubMed Central, Google Scholar made using such terms as; urinary schistosomiasis, Bilharziasis, urogenital complications, medical treatment, ablative and reconstructive surgeries yielded 125 suitable articles which were incorporated into this review.*

Conclusion*: Urinary schistosomiasis is an endemic parasitic disease in many topical countries of Africa and south-west Asia and afflicts millions of subjects. Heavy infestation not adequately treated may result in serious complications that are organ-specific and affect the urogenital system with attendant morbidity and mortality. Preventive strategies are mandatory to reduce these sequalae of the parasitic disease.*

Key words*: Urinary schistosomiasis, pathogenesis, complications, treatment and prevention*

I. Introduction

Genitourinary Schistosomiasis remains a worldwide parasitic disease caused by Schistosoma haematobium[1] which remains one of the most extensively studied parasitic diseases afflicting over 200 million people all over the World. The disease spreads across tropical Africa, the Middle East, and South-West Asia with the majority of the population resident in developing countries causing attendant socio-economic and public health

consequences. Schistosomiasis exists in two forms-the intestinal and the genitourinary with the intestinal form occurring as a result of any of the five main species of the Schistosoma however the genitourinary system is only affected by the Schistosoma haematobium.[2]

Though the majority of urinary Schistosomiasis may result in spontaneous regression or mild consequences, about 10% may progress to pathologic disease states causing deleterious effects on the bladder function, ureters and kidneys and this is associated with high morbidity and mortality depending on the intensity of the infection, thus the severity of the resultant disease especially in children in whom considerable damage may be done to the urinary tract.[3, 4]

This high morbidity and mortality are prevalent in the developing countries of Africa, South America, the Caribbean, the Middle East, and Asia due to the high endemicity of the parasite whereas occurrences in the more developed and industrialized nations of the world are a result travelling for tourism and from subjects immigrating from these areas with high endemicity.[1, 5]This current trend of exportation of urinary schistosomiasis from the high endemic developing sub-Saharan countries to the more developed temperate regions of the world makes this review necessary to alert physicians in these countries of the manifestations and clinical complications of urinary schistosomiasis. A high degree of morbidity and mortality in third world countries especially Africa is associated with infestation by Schistosomiasis (Bilharziasis).[6]There is, therefore, a need to initiate programs that seek to address mass control of the disease to eradicate the disease.

II. Methodology

Electronic literature searches on PubMed Central, Google Scholar made using such terms as; urinary schistosomiasis, Bilharziasis, urogenital complications, medical treatment, ablative and reconstructive surgeries yielded 125 suitable articles which were incorporated into this review. All articles were open access and in English published between 1967 and 2022.The author also included other relevant publications in those searches.

Epidemiology of schistosomiasis

Schistosomiasis is the second most common type of neglected tropical disease, an entity that comprises a group of chronic, disfiguring conditions that occur among poor, rural community dwellers and exert significant economic and public health impact.[7, 8]Eighty-five per cent of the over 207 million people with Schistosomiasis reside in Africa with its prevalence due to lack of access to safe drinking water and inadequate sanitation.[2] Specific country of schistosomiasis hyper-endemicity in Africa includes Nigeria, Tanzania, Egypt.[6, 9-11]This disease affects mainly young children and has a great impact on the economy, level of poverty and public health in the affected nations.[11-13] Schistosomiasis exists in two forms-the intestinal and the genitourinary with the intestinal form occurring as a result of any of the five main species of the Schistosoma however the genitourinary system is only affected by the Schistosoma haematobium.[2]

The blood fluke of the genus schistosomiasis is responsible for the Schistosomiasis infection with at least five of such trematode species linked to the infection of humans. These species are Schistosoma haematobium, S intercalatum, Schistosoma japonicum, Schistosoma mansoni, Schistosoma mekongi[14] and Schistosoma guineensis (the Lower Guinea species).[4, 6] Human Schistosomiasis can occur following water containing cercariae.

Schistosoma haematobium is the only species that infect the genitourinary system resulting in a barrage of clinical symptoms and signs.

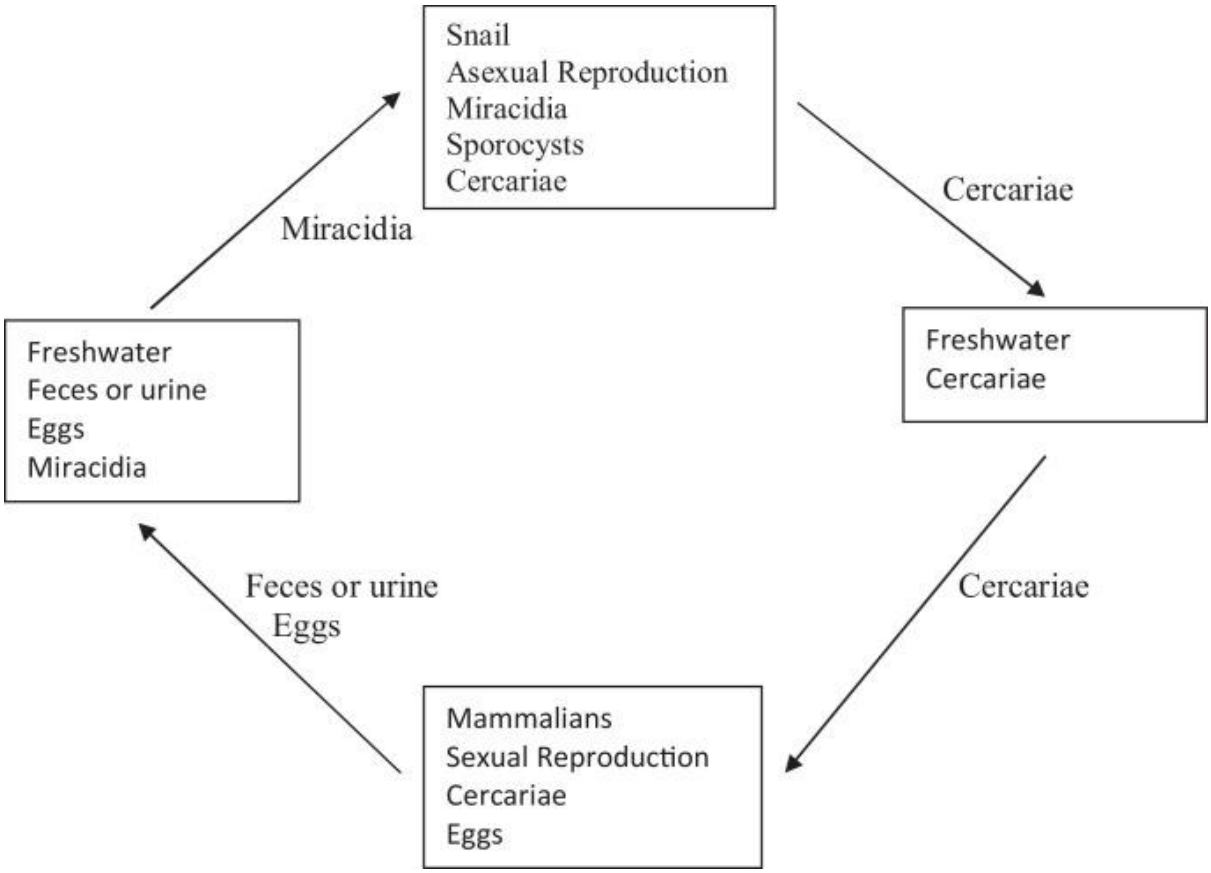

Figure 1: Schistosomiasis life cycle[14]-Asexual reproduction in Snails and Sexual reproduction in Mammals.

Trematode specie	*Snail Host (Genus)*	*Number of Cercariae shed daily*
S.haematobium	*Bulinus*	*About 200*
S.japanicum	*Oncomelania*	*15 -160*
S.mekongi	*Neutricula*	
S.mansoni	*Biomphalaria*	*250-600*
S.intercalatum		

Mammalian Hosts.

The **Cercariae** shed their forked tail once they enter the human skin forming **schistosomula.** Resultant migration of the schistosomula throughout the body's tissues through the blood circulation into **schistosomules** and **adult worms**. The adult worms consist of males and females having ZZ and ZW chromosome pairs respectively.

Location of the adult worms in Humans.

Trematode specie	*Location*
S.haematobium	*Bladder, ureters, rectal venules.*
S.japanicum	*Frequently in the small intestine*
S.mansoni	*Large or small intestine.*

Taxonomy of schistosoma haematobium

These Schistosomes are blood-dwelling parasitic fluke worms which belong to the genus Schistosoma; family, Schistosomatidae; order, Digenea; class, Trematoda; phylum, Plathyhelminths; and kingdom, Animalia.
The Schistosomes are the trematodes of most pathologic importance however they are not hermaphroditic having separate sexes. The adult worms measure 1-2cm in length with a cylindrical body featuring two-terminal suckers, a complex tegument, a blind digestive tract, and reproductive organs. The body of the male forms a groove or gynaecophoric channel which holds the longer and thinner female. The couples are permanently embraced with the schistosomes living within the perivesical (S. haematobium) or mesenteric (as applies to other species) venous plexus. The Schistosomes feeds on blood particles via anaerobic glycolysis.[15]

Life cycle of schistosoma haematobium

The female worm produces about hundreds of eggs per day which measure 144 X 58 µm characterized by a terminal spine that penetrates through the bladder wall excreting with the urine from the bladder. Each ovum contains miracidium (ciliated ova) which secretes proteolytic enzymes which helps the eggs to migrate into the bladder lumen. With close to half of the eggs produced not able to reach the vesical lumen being carried away with the bloodstream or trapped in the tissues. The eggs retained is known to provoke a granulomatous inflammatory response implicated in the pathology in the human host. The eggs that are excreted hatch when they come in contact with water releasing miracidium however they remain viable for up to 48 hours being able to locate a suitable freshwater snail host specifically Bulinus spp. for the S. haematobium. The snail species is attracted to the miracidium by external stimuli like light and small-derived chemicals. Asexual multiplication takes place in the snail with larvae (sporocysts) multiplying in generations eventually these Sporocysts produce large numbers of Cercariae (infective larvae with a typical bifurcated tail). The Cercariae leave the snail at a rate of thousands per day after several weeks with the shed of the Cercariae lasting months. The survival of the Cercariae may last up to 72 hours using water turbulence chemicals derived from the skin to locate the human host and within 3-5 minutes attach themselves and penetrate the human skin. As soon they lose their tail, the schistosomule (young parasites) migrate with the bloodstream through the lungs to the liver, maturing into adult worms in the portal vein and then mate.
The paired worms migrate against the bloodstream to reach the perivesicular veins producing eggs 4-7 weeks after infection and continuing throughout their adult life. The adult worm spans an average of 3-5 years lasting up to 30 years.[16] Once a person is infected, an average of 10s-1000s of worms is harboured.[17]

Pathogenesis and morbidity of urinary schistosomiasis

The clinical features and organ-specific complications of urinary schistosomiasis arise as a result of the body's response to the eggs trapped in the tissues of the urogenital system.[18] The presence of the eggs induces a granulomatous host immune response involving the lymphocytes, eosinophils and activated macrophages and the magnitude of this depends on the host genetics, the intensity of the infection, in utero sensitization to schistosomal antigens and the co-infection status.[19, 20]
Schistosoma haematobium eggs deposited in the urinary bladder evoke an immediate immune response from the host within 24 hours and this involves local and recruited innate immune cells leading to induction T helper cells, tumour necrosis factor, interleukins and other cytokines.[21-23]

Clinical manifestations of urinary schistosomiasis

Clinical presentation of urinary schistosomiasis depends on the stage of the infection, urogenital organ involvement, subject's immune status determined by previous exposure to the schistosomal antigens as occurs in subjects living in endemic regions as opposed to those coming from endemic regions of the world. The disease may present as acute, subacute or chronic disease and complications as a result of damage to the urogenital organs.

Acute schistosomiasis occurs in the early stages of skin penetration, invasion and migration usually occur in non-immune such as in travelers or immigrants from schistosoma non-endemic to endemic regions due to the absence of prior exposure to the schistosoma antigen.[24, 25]

This occurs weeks to months after infection and may manifest as dermatitis (swimmer's itch)resulting from cercariae penetration of the skin.[26-29] The bronchopulmonary manifestation of acute schistosomiasis is due to the passage of the parasites through the lungs.[30, 31]Anotherclinical presentation of acute schistosomiasis is the Katayama syndrome which represents a formof hypersensitivity reaction to the migratory schistosomules and the time of its manifestationcoincides larval maturation, migration, early oviposition and is characterized by malaise, high-grade fever, myalgia, headache and abdominal pains.[32-34] This syndrome has been reportednot only with infestation with Schistosoma haematobium but also with S. mansoni and Sintercalatum.[26]Markers of inflammatory response such as erythrocyte sedimentation rate, C- reactive protein, serum amyloid A may be moderately elevation though there may be marked eosinophilia that may persist long after anti-helminthic and anti-inflammatory therapy.[34]

Subacute manifestations of urinary schistosomiasis usually present after worm maturity and deposition of the dead worms and eggs in the urinary bladder and the symptoms are due to the formation of granuloma around the dead worms and eggs.[35] Coalescence of the granulomas forms the characteristic pseudotubercles pathologic feature of the early disease. The ensuing urinary wall and mucosal hypertrophy in addition to the congregation of the pseudotubercles give rise to the ulcerating polypoid lesion that bleeds easily.[36, 37] The granuloma deposition in the bladder causes urinary frequency, painful micturition, suprapubic pain, gross haematuria and obstructive uropathies.[24]

Chronic urinary schistosomiasis may appear many years after the initial symptomatic or asymptomatic disease especially in subjects residing in highly endemic areas.[38]

In these subjects residing in schistosoma-endemic regions, the infection may be acquired in early childhood as early as age five years and the intensity of the infection may increase in intensity due to repeated exposure through contact with infested fresh water sources and after this period, the infection persists at low levels throughout adult life.[39]

The pathological lesions that occur in chronic urinary schistosomiasis result from the immunological responses to the eggs that have been trapped in the genitourinary tract which are initially of granulomatous response but with time progress to focal fibrosis in the affected organs.[40] Primary target organs for urinary schistosomiasis include the bladder, lower ureters, seminal vesicles and less frequently, the prostate, vas deferens, and the female genital system.[35]

R**adiological findings**

The pathological sequalae in urinary schistosomiasis may be demonstrated using different imaging modalities such as ultrasound, plain and contrast X-rays and computerized tomographic scans.[41]

Ultrasound continues to play an important role in the diagnosis of the complications of the disease as well as in follow up of patients with urinary schistosomiasis.[42] The advantages of ultrasound include wide availability, is inexpensive, does involve the use of ionizing radiation and does not require special structures for installation and use. Urinary bladder ultrasound findings depend on the stage of the disease. In the acute phase wave-like bladder enlargement, focal pseudopolypoid lesions and increased vascularity while in the chronic state may show increased wall thickness, wall calcification, endoluminal calculi, bladder masses, significant post-void residual urine due to bladder stenosis from contracture as well as upper tract changes manifested as hydro uretero-nephrosis and features of female genital schistosomiasis**.**[43-45]

In the past, intravenous urography was the diagnostic modality of choice in imaging of the urinary tract in chronic urinary schistosomiasis, however, in the recent times, this has been supplanted by cross sectional

imaging techniques such as computerized tomography (CT) scan magnetic resonance imaging (MRI) which are able to detect obstructing radiolucent ureteral calculi, demonstrate a better detained anatomy of the renal parenchyma, the ureters, the urinary bladder, surrounding spaces as well as assessment of the urothelial wall.[46-50] MRI, however, is indicated in pregnant patients, those with contrast allergies and it offers better tissue characterization.[50]

Imaging of the Ureter in Genito-urinary Schistosomiasis

The ureters and the bladder are the main parts of the urogenital system involved in genito-urinary schistosomiasis however the kidneys may show features rather at a later stage of the disease.[2]Schistosomiasis involves about 2/3rd of cases[47, 51] with a persistent filling of the distal ureteral segment and later distal ureteral dilatation being the earliest visible findings on the urogram. At an early stage, the dilatation of the ureter arises from ureteral dysfunction as against the tissue damage with majority unilateral. Later on, in the disease progression, ureteral strictures may arise from tissue healing as a result of ureteral fibrosis with a majority of the earlier ureteric strictures arising from the intravesical segment of the ureter, followed closely by the part 2-3 cm above the ureteric orifice. The ureteric stricture may extend involving the entire length of the ureter following the extension of the fibrosis above and below the stenotic segment of the ureter.[42, 52]

Cystoscopic findings of schistosomiasis of the bladder

Findings in the urinary bladder in urinary schistosomiasis depend on the changes induced by the presence of the ova of the parasite which vary on the stage of the disease. These include mucosa congestion, hyperaemia, submucosal haemorrhage, ulceration, cystic lesions, pale mucosal mucosa patches (sandy), submucosal pseudotubercles often located around the trigone and posterior bladder wall, polypoid lesions, granulomas, ulcer (SCC) with phosphate encrustations and sandy patches and lastly fungating malignant mas (SCC).[35, 37, 53, 54]

Genitourinary complications of schistosomiasis

Schistosomiasis of the urinary tract may regress spontaneously or result in mild consequences however 10% of the patients may eventually progress to pathologic states of the disease. These pathologic states may involve the bladder, ureters, and kidneys resulting in death from renal impairment or cancer bladder.[55] Factors that affect the extent of complications include the tissue egg load, occurrence of bilharzial infestation in terms of duration and frequency, appropriate treatment and the influence of secondary microbial infection alongside the presence of atrophic, proliferating epithelial lesions of metaplastic origin occurring in the bladder and the distal ureters. The Schistosomal parasite rarely invades the kidneys and genitals.[56]

Male genital schistosomiasis

Male genital schistosomiasis is the manifestation of urinary schistosomiasis demonstrated by pathological changes and presences of the ova in the male genital organs or reproductive fluids.[57] Involvement of the male genital organs occurs following the migration of the worms to these organs after repeated parasitic infection. It may involve the prostate, seminal vesicles, spermatic cord and epididymis. The resultant severe fibrosis due to the immunologic response to the presence of the ova may lead to calcification with the formation of a firm, nodular palpable seminal vesicle. The disease may be diagnosed as an incidental finding or as misdiagnosis of testicular tumour, prostate cancer and part of investigation for may factor infertility.[58-61]Male factor infertility as a complication of schistosomiasis may occur as a pretesticular phenomenon due to alteration in the hypothalamic-gonadal axis; from testicular damage from inflammation or post-testicular fibrosis of the ejaculatory ducts and destruction of the accessory male sex organs including the prostate, seminal vesicles and the resultant azoospermia and oligospermia .[61]

Migration of the worms following repeated Schistosomiasis and in severe forms to other organs like the prostate, seminal vesicles, and the intra-scrotal structures however the testis is known to have remarkable

immunity against the invasion by the bilharzia. The resultant severe fibrosis arose from the repeated infiltrations by the Schistosoma with marked fibrosis causing marked calcification resulting in a firm, nodular and palpable seminal vesicle.[62, 63]

Female genital schistosomiasis

Female genital schistosomiasis (FGS) is the manifestation of bilharziasis in female subjects characterized by the presence of the ova or worms in the upper and lower genital tract.[64] FGS has a high prevalence in bilharzia endemic regions I sub-Saharan Africa and is due to infection with S. haematabium.[65, 66] In such regions, FGS is a common cause of gynaecologic disease and the genital lesions may result in damages that predispose the subjects to sexually transmitted infections and the human immunodeficiency virus infection.[25, 67]

Genital organs affected by FGS include the ovaries, fallopian tubes, uterus, vagina and the vulva.[64, 68] Clinical signs and symptoms of FGS are not specific but may be related to the affected genital organ and may consist of irregular menstruation, odorous vaginal discharge, dyspareunia, post-coital bleeding, vulval-vaginal oedema, cervical erosions, recurrent spontaneous abortions, ectopic pregnancy, tubal abortion, anaemia due to chronic blood loss, vesicovaginal fistula, primary or secondary infertility.[64, 65, 69]

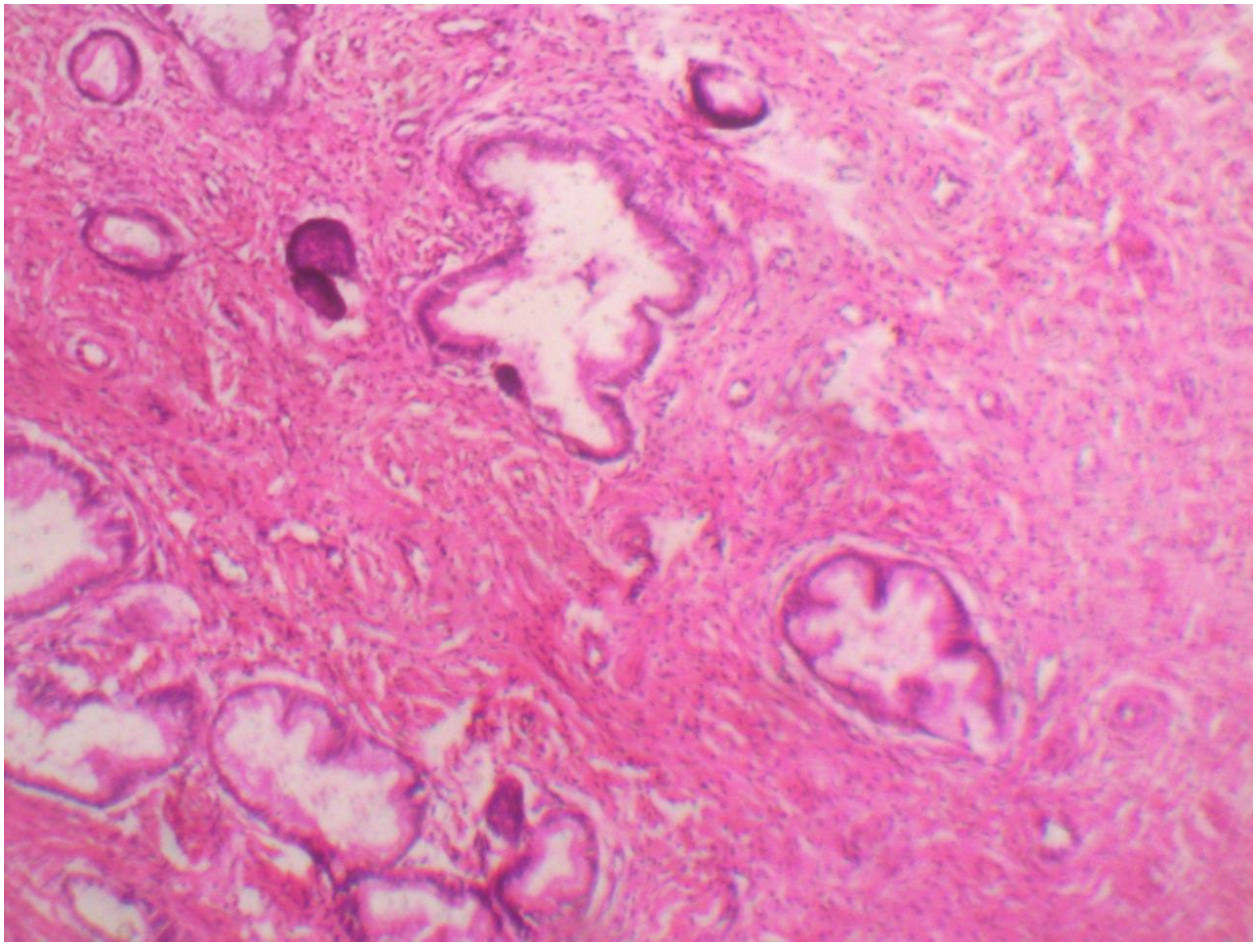

Figure 1: Schistosoma Cervix

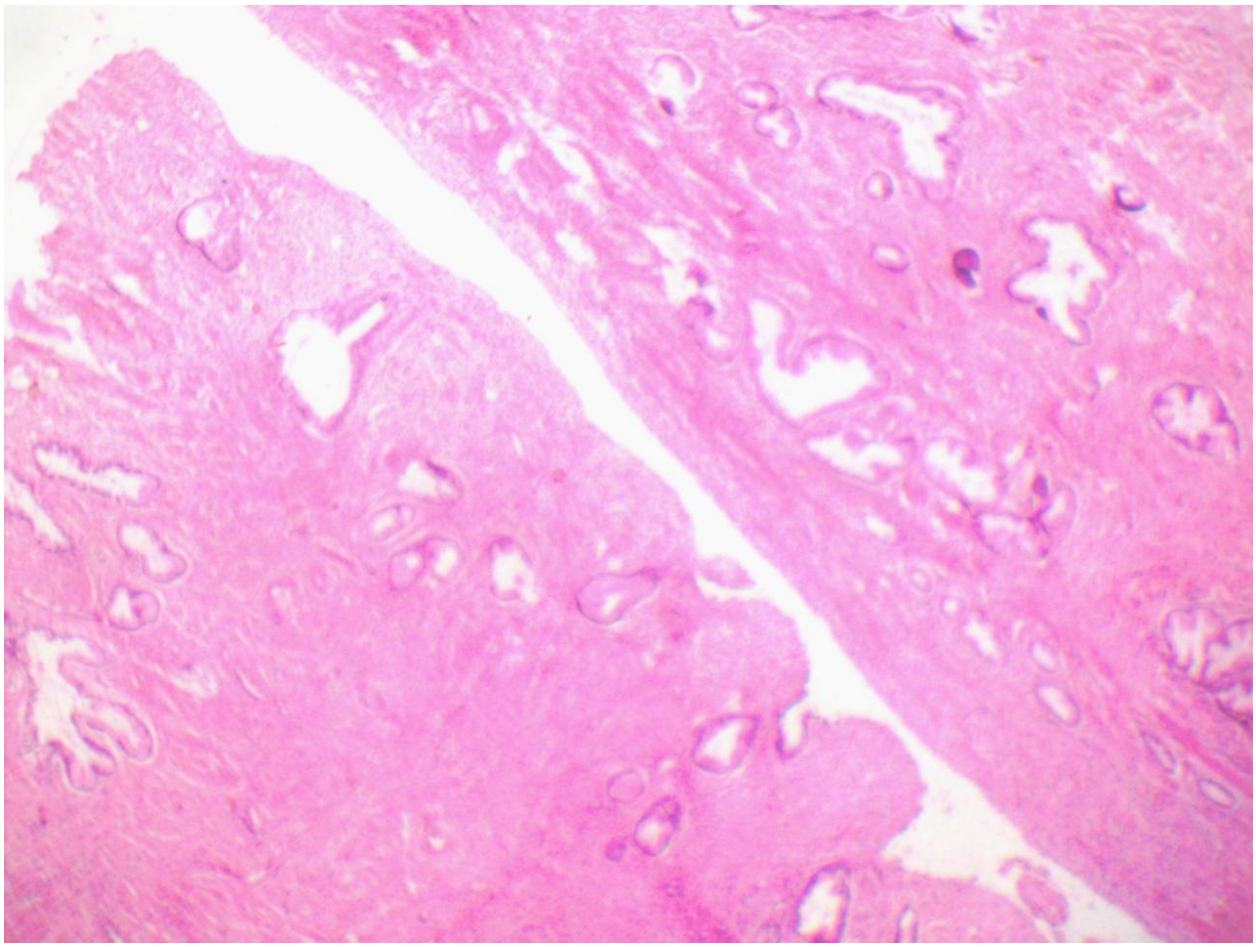

Figure 2: Schistosoma Endocervix

Chronic Benign Schistosoma bladder lesions

The urinary system is one of the primary target genitourinary organs for schistosomiasis. It affects all the layers of the bladder resulting in either an atrophic or hyperplastic pattern. The lesions may be mucosal or submucosal in origin with differing patterns. The mucosal lesions include ground-glass ischemic changes, cystitis-cystica, calcifications, ulcers and leukoplakia whereas the submucosal lesions include fibrosis, calcifications, nodules and granulomata.[2] Ultrasonographic appearance of chronic urinary schistosomiasis may include diffuse thickening, calcification of the bladder wall, parietal nodes, irregularities of mucous profile such as hyperplasia focal nodular pseudopolypoid lesions.[70, 71] Degeneration in the muscular layer, bladder neck fibrosis and contracted bladder are amongst the muscular lesions seen in Schistosomiasis.

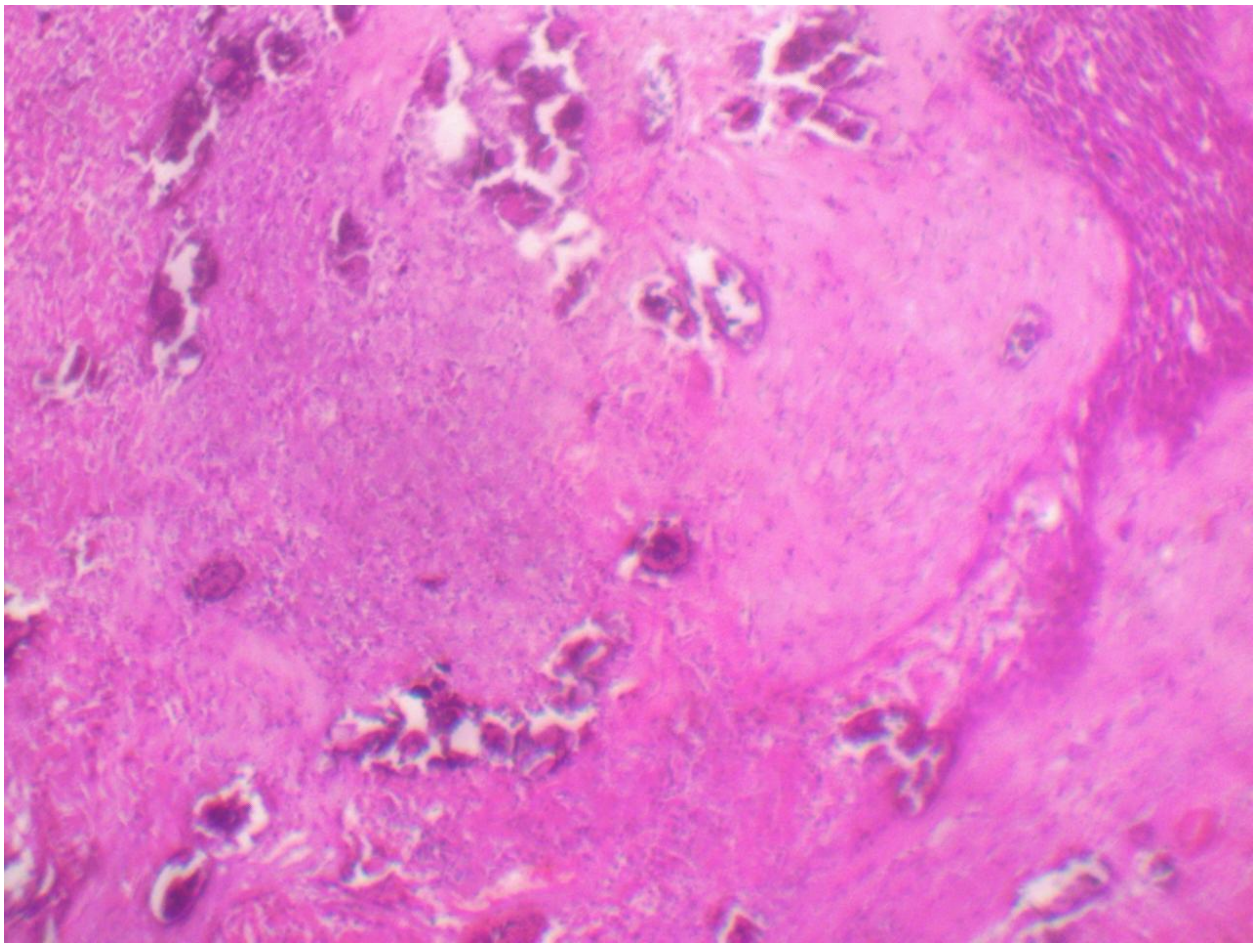

Figure 3: Schistosoma Bladder

Obstructive Nephropathy and renal failure

Renal injury in patients with schistosomiasis haematobium infection occurs by a variety of mechanisms including the body response to the deposition of circulating antigen-antibody complexes in the glomerular capillaries and basement membrane and the resultant glomerular injury.[72] The induced immunologic response may cause glomerulonephritis., proteinuria, nephrotic syndrome and subsequent renal failure.[73, 74]The second mechanism of nephropathy due to urinary schistosomiasis arise from the bladder trigonal involvement, infection, oedema, configure changes, functional obstruction, ureterovesical obstruction and upper tract changes. The unrelieved obstruction and reflux may result in infection, chronic interstitial nephritis with fibrosis, and impaired renal function.[75]

However, following the administration of anti-bilharzial medications, they may be a regression of the acute and early chronic lesions, but this treatment may not stop the irreversibility of the chronic sequelae of end-stage renal failure.

Bladder cancer associated with Schistosomiasis

The predominant type of cancer associated with Schistosoma infested bladder is squamous cell carcinoma (SCC).[76] The incidence of SCC following Schistosomal bladder was highest in Egypt when compared with other countries in the middle east and Africa however the low incidence in other countries was due to the less endemicity and severity of the Schistosoma infection. SCC constitutes 65.1% of histologically confirmed cases of bladder cancers in Sokoto, Northwestern Nigeria with 50% of the squamous cell carcinoma revealing chronic

urinary schistosomiasis.[77] The prevalence of Schistosoma associatedSCC bladder cancer in Egypt is decreasing over time which is not unrelated to the aggressive measures at combating the infestation of Schistosomiasis.[78]

Bladder cancer and urinary schistosomiasis have an intricate relationship following several factors etiologically harmonized which includes chronic irritation of the bladder mucosa by the Schistosoma ova, recurrent bacterial infection, toxins released from the carcinogens produced by the miracidium or worms and raised metabolites from tryptophan, nitrates, serotonin and urinary beta-glucuronidase enzyme. The bacteria involved release free carcinogenic products like nitrosamine from the urine nitrates and secondary amines.[55]

The bladder cancer from non-Schistosoma patients differs from that of schistosomiasis as its entity presents as a single bulky, nodular, invasive low-grade hyperkeratotic SCC and mostly local malignant. The origin can be from any part of the bladder except the trigone constituting 2% of cases.[79, 80] The nodular fungating type constitutes 85% of cases with the less common forms constituting 3.6%. The less common forms consisting of ulcerative, diffuse, infiltrative and verrucous are rarely peculiar to Schistosoma SCC and do not have any tendency to spread to lymph nodes or distant organs.[55]

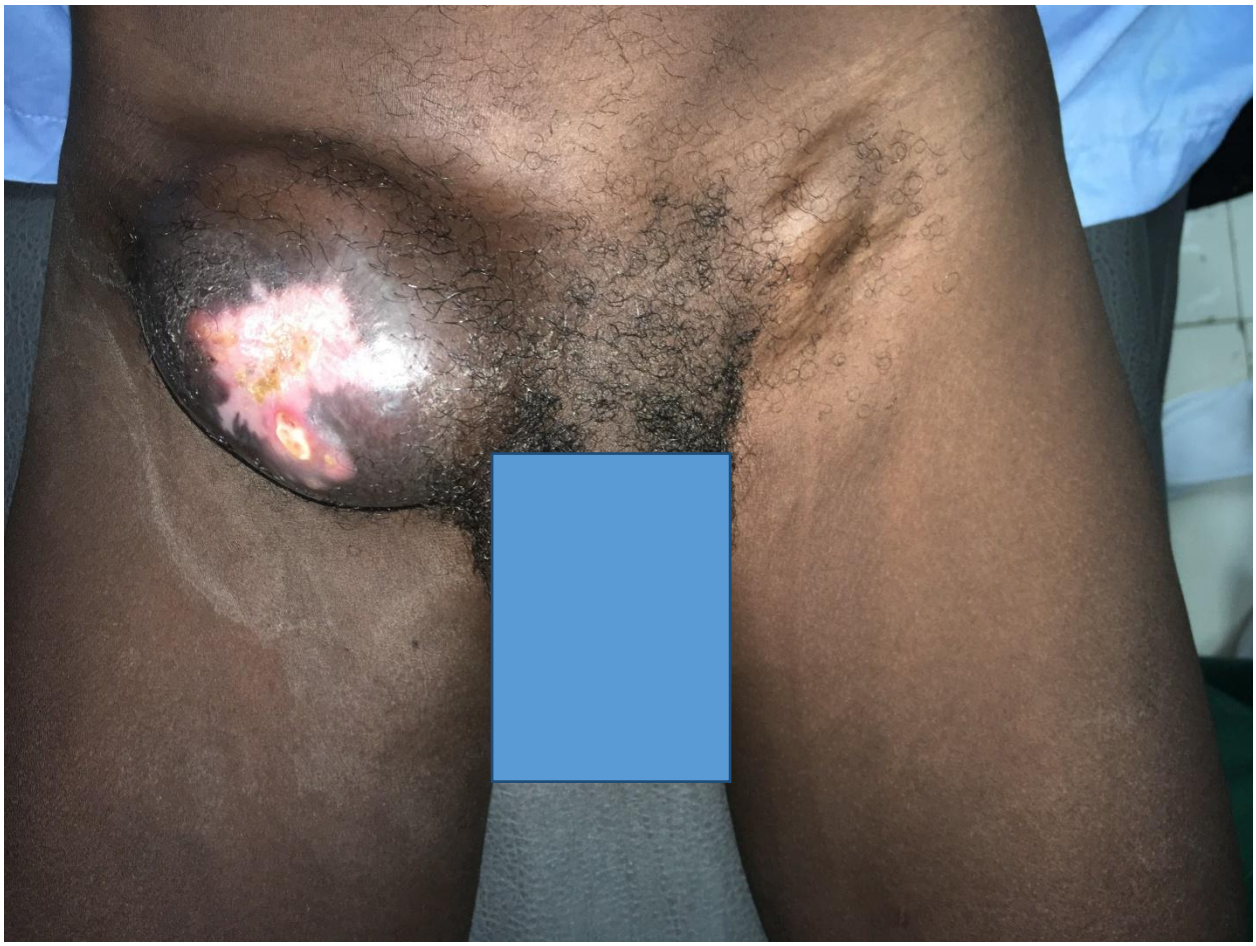

Figure 4: Ulcerated Right groin lymph nodes and Left Groin nodes on a background Metastatic Squamous CellCarcinoma.

Schistosoma ureteric lesions

Ureteral involvement in schistosomiasis is important for the renal complications discussed above and the lower ureters are usually the commonest sites.[81]This location corresponds to the anastomotic sites between the inferior mesenteric and the peri-ureteric and the perivesical veins which are the routes through which Schistosoma

haematobium migrate into the urinary tract.[75] The ureteric lesions and the fibrotic pathology resemble those of the bladder with the resultant partial ureteric obstruction.[82]The lesions of the ureters include cystica, calcinosa, glandularis and fibrosis of the ureter which may progress to fibrosis with associated mucosal degeneration, obliterated ureteritis and consequently strictures. The final pathway of the chronic Schistosoma ureter is that of a thick rigid ureter appearing like a pipe-stem, strictured ureter, dilated atonic ureter and in some scenarios, a combination may present.[56] Renal dysfunctions may arise from ureteric obstruction following ureteric tortuosity, ureteric kinking resulting in backpressure. The common presentation is that of a dilated ureter in the pelvic ureter while the dilatation of a non-obstructed ureter is a result of ureteric muscular and neural degeneration or following vesicoureteral reflux.[83] SCC associated ureteric strictures rarely develop from repeated Schistosoma infestation and microbial infection.[55]

Schistosoma related urinary stones

Previous studies carried out in Egypt did not show any statistically significant difference in urinary stone disease amongst Schistosoma and non-Schistosoma infested patients.[84] The study from Sudan also corroborated the earlier mentioned report.[85] The aforementioned confirms the non-relationship between urinary stones and schistosomiasis. The higher frequency of ureteric calculi among other urinary stones arises due to the peculiarities of the Schistosoma ureter as a primary site of stone formation as the migrating stones could be arrested.[56] Obstruction and urinary infection may result in giant stone formation at the bladder and ureter.[84]

Diagnosis of genitourinary schistosoma complications

The diagnosis of urinary schistosomiasis both in the early stages and its later genito-urinary complications involves the application of direct parasitological tests, immunological tests, DNA and RNA- based techniques, measurement of cytokines, parasite metabolic products levels, Schistosoma molecular markers, histopathological tissues assays as well as the use of radiological imaging methods.[43, 86] Identification of calcified bilharzial ova may be demonstrated in histopathological specimens of the cervix, prostate, testis, seminiferous tubules, and semen.[58, 64, 65, 87, 88] In addition, a thorough clinical urologic examination may include DRE and bimanual examination, digital urologic imaging, cystoscopy and biopsy of the tissue. Abdominal ultrasound may reveal echogenicity in the bladder growth on a background hypertrophic lesion or thickening of the wall of the bladder. Plain abdominal radiograph (KUB) may reveal stones or a characteristic Schistosoma linear calcification in the bladder. Contrast studies may show filling defects or a contracted bladder.[89]

The Schistosoma granuloma may present clinically as total haematuria with repeated attacks commonly seen. Schistosoma nodules may mimic bladder carcinoma following cystoscopy and on Intravenous urography especially when they acquire large dimensions.[55] Follow-up is mandatory following the visualization of leukoplakia on cystoscopy.[56]

Some patients may present with chronic bilharzial ulcers especially among those in the 3rd and 4th decade often associated with post-micturition pain and sometimes haematuria. The confirmatory diagnosis is cystoscopy.[90]

Difficulty in initiating voiding, straining, and weak interrupted stream in young males may suggest bladder neck obstruction (BNO)[91] however the diagnosis can only be made by urodynamic pressure-flow study alongside imaging.

The SCC of the bladder seen in our part of the World differs from what is obtainable in the Western World as its presentation is that of painful micturition, frequency, and haematuria classically symptoms of cystitis. Though urogram may reveal an irregular filling defect, diagnosis depends on cystoscopy and biopsy, careful bimanual examination under anaesthesia.[62, 92] The role of urine cytology is significant as a diagnostic tool for SCC in patients with schistosomiasis.[93] There is often associated clinical error and a high likelihood for underestimation associated with clinical staging.[94] Sadly, the majority of the patients present for treatment at an advanced stage with a quarter being inoperable.[94, 95]

The diagnosis of ureteric strictures largely depends on the visualization of the urinary tract through imaging such as CT urography or magnetic urography where facilities are available and in selected cases use of diuretic renography alongside washout technique and perfusion pressure-flow studies.[55]

Interestingly, Schistosoma involvement of the prostate and seminal vesicles have no known complications with haemospermia rarely presenting in male patients with genitourinary schistosomiasis.[96]

Management of schistosomiasis and urogenital complications

Management of urogenital schistosomiasis involves treatment of the acute and sub-acute forms of the disease, the complications and the measures that aim at preventing the initiation of the disease. Drug therapy remains the mainstay of the treatment of urogenital schistosomiasis especially in the acute and subacute stages, however, in severely damaged organs and most commonly, when clinically misdiagnosed for malignancy aggressive extirpative surgeries such as radical orchidectomy, salpingo-oophorectomy and hysterectomy have been done.[59, 97]

Drug treatment of urogenital schistosomiasis

Praziquantel is the most widely used drug both in the treatment and in the control of urinary schistosomiasis as part of the de-worming program in endemic locations.[98, 99] It has demonstrated significant efficacy in the cure, reduction of egg counts as well as a decrease in symptoms of haematuria and proteinuria in the affected patient population.[100]

Several treatment regimens have been in the use of Praziquantel in the treatment of urinary schistosomiasis including a single dose of 40mg/kg or as repeated,3-monthly 60mg/kg re-dosing schedules as well as in the combination of the anti-malaria agent; artemisinin.[101-104]

Though Praziquantel has demonstrated admirable efficacy and safety both in adults and children, it has however been associated with minor, self-limited adverse effects such as abdominal pain, diarrhea, dizziness, nausea, vomiting and rashes.[105]

Another drug that may be used in the treatment of urinary schistosomiasis is metrifonate, an organophosphate that kills the parasites by inhibition of the cholinesterase enzyme usually administered at three bi-weekly intervals of 5mg/kg though this dosing schedule makes it not readily desirable in mobile populations in endemic and is thus inferior to praziquantel.[98, 106]

Surgical treatment of urogenital schistosomiasis complications

Surgical treatment options for urogenital schistosomiasis range from endoscopic minimally invasive procedures, reconstructive and ablative surgeries depending on organ involvement, the severity of organ destruction and biochemical derangement.[98]

Endoscopic bladder procedures in schistosomiasis include transurethral resection of bladder neck -stenosis, for removal of polypoid lesions to obtain tumour histology and to achieve relief of disturbing irritative lower urinary tract symptoms and reduce gross haematuria[107], endoscopic ureteral procedures in bilharziasis are indicated in short segment strictures and include ureteral meatotomy, endoureterotomy, percutaneous antegrade balloon dilatation.[108, 109]

Urinary schistosomiasis has a predilection for the distal ureters, urinary bladder and the pathologic ureteric strictures result in obstruction and the consequent obstructive nephropathy.[110, 111]Reconstructive surgical treatments for urinary schistosomiasis include procedures that aim to relieve the effects of ureteric and bladder fibrosis, with the resultant obstructive nephropathy and troublesome irritative voiding symptoms from the contracted bladder.

The surgical technique adopted for ureteral reconstructive surgeries depend on the length of the affected segment and these may be carried as open, laparoscopic or robotic-assisted procedures in practices where the equipment and expertise are available.[112-114] These include ureteroneocystostomy, Boari flap, ureteroureterostomy, psoas hitch and ileal replacement.[111, 115, 116]

Prevention of genitourinary schistosomiasis

Prevention and control of urinary schistosomiasis with the consequent urogenital complications is based on multiple approaches such as mass drug administration, measures that interrupt the transmission of the parasites, health education program as well as the probable administration of vaccines.[117-120] A couple of pharmacological agents have been administered as part of preventive and control measures for urinary schistosomiasis. Praziquantel is the most commonly used chemotherapeutic agent in the prevention and control of urinary schistosomiasis as well as other drugs such as artesunate and artemether.[99, 121] Though use of mas drug administration has the objective of reducing disease morbidity and mortality as a result of limiting transmission, reducing prevalence in the population, it suffers the drawback caused by non- coverage of the whole population especially non-enrolled school children at the time of the program.[122]

Effective non-pharmacological schistosoma control strategy should be an integrated as a multi-specialty program consisting of population health education, water hygiene, and sanitation, snail elimination and this must involve community participation and acceptance.[119, 123-125]

III. Conclusion

Urinary schistosomiasis is an endemic parasitic disease in many topical countries of Africa and south-west Asia and afflicts millions of subjects. Though mild infestations could result in sub- clinical symptoms that may regress spontaneously, however, heavy infestation not adequately treated could result in serious complications that are organ-specific affecting the urogenital system with attendant morbidity and mortality such as chronic renal failure and bladder cancer. The complications of urogenital schistosomiasis may be surgically treated by an array of ablative and reconstruction procedures.

Preventive strategies comprising of environmental sanitation, water, snail control measures and pharmacologic treatment are mandatory to reduce the severe sequalae of these parasitic disease.

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