Introduction

Schistosomiasis is a major infectious disease of public health and socioeconomic importance in the developing world. Schistosomiasis also known as bilharzia, bilharziosis or snail fever is a parasitic disease caused by several species of fluke of the genus *Schistosoma* . This disease is most commonly found in Asia, Africa, and South America, especially in areas where the water contains numerous freshwater snails, which may carry the parasite. Both *S. haematobium* and *S. mansoni* are present in Sudan, a war-torn country with a population of ≈30 million persons and one of the world most underdeveloped regions. Risk for schistosomiasis in Sudan is widespread, especially in the major irrigation systems in the Gezira area between the Blue and White Nile Rivers. The disease affects many people in developing countries, particularly children who may acquire the disease by swimming or playing in infected water. Although it has a low mortality rate, schistosomiasis often is a chronic illness that can damage internal organs and in children impairs growth and cognitive development. The urinary form of schistosomiasis is associated with increased risks for bladder cancer in adults. Schistosomiasis is the second most socioeconomically devastating parasitic disease after malaria (Carter center, 2008).

Schistosomiasis is a chronic disease and infections are subclinical symptomatic, with mild anemia and malnutrition being common in endemic areas. Acute schistosomiasis)Katayama's fever (may occur weeks after the

initial infection (Girges, 1934).

Chapter one

Literature review

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Literature review

1.1 Historical background :

The earliest description of schistosomiasis is found in the Papyrus Ebers of ancient Egypt. The ancient Assyro-Babylonian literature alludes to a worm disease associated with urinary bleeding. It was not, however, until the nineteenth century that a systematic attempt was made to understand the basic life-cycle and pathogenesis of disease. The German pathologist Theodor Bilharz is credited with the first description of the adult worm, which he found in the portal vein of a young man at autopsy (cited by Beaver 1984). In retrospect, it is unclear whether Bilharz was describing *S. mansoni* or *S. haematobium*, since he described eggs with a terminal spine. He also described the characteristic pathologic changes and clinical features of schistosomiasis.

Schistosoma haematobium infection has been known to occur since earliest times, and schistosome eggs have been recovered from both China and Egyptian mummies in the Nile valley for several millennia (Beaver *et al*., 1984) showing that the infection was present in both of these early civilization of mankind. Haematuria one of its cardinal signs, was described in the gynaecological papyrus of Kahun, one of the oldest papyrus

fragments found in Egypt dating back to the year 1900 B.C (Beaver *et al* 1984). It was first noted as early as 1910 by Sir Armad Ruffer, who found calcified eggs in the kidneys of two mummies of the twentieth dynasty. Beaver *et al.*, (1984) mentioned that the symptoms of the disease were frequent among the soldiers of Napoleon during the Egyptian campaign (1799-1801). The worms were first recovery in 1851 by Theodor Bilharz, during his working in Qasr El Aini hospital, in Cairo, in the mesenteric veins of a cadaver at post-mortem examination and named it *Distoma haematobium*. Shortly, thereafter, he demonstrated them to be the cause of haematuria in native laborers who were discharging terminal spined egg of the fluke in the urine. In 1864, Harley reported the organism from South Africa under the name Bilharzia capensis (Beaver *et al*., 1984). In the Far East, infection with related species *S. japonicum* was also recognized, where it was know as Katayama disease, characterized by enlargement of the liver and spleen, abnormal feelings of hunger and bloody diarrhea and fever so called (Katayama Fever)

1.1.1 Schistosoma haematobium:

S. haematobium is an important digenetic trematode, which is found in Africa and the Middle East. It is a major agent of schistosomiasis; more specifically, it is associated with urinary schistosomiasis. The inter-mediate host is the fresh water snail of the genus *Bulinus* (Leutscher *et al*. 2005).

1.2 Classification

S.haematobium is classified according to Soulsby (1986) as follows:

Phylum : *Platy helminthes*

Class : *Trematoda*

Subclass : *Digenea*

Order : *Protostomata*

Suborder : *Strigeata*

Family : *Schistosomatoidea*

Genu : *Schistosoma*

Species : *haematopium*

1.3 Morphology and life cycle:

Schistosoma are long-lived worms, having life span of 20 to 30 years, Adult worm live in the pelvic venous plexus-vesicle, prostatic and uterine plexuses of veins. The three species of adult worm resemble each other closely, have separate sexes, *S.haematobium* male is short and cylindrical, measuring 10 – 15 x o.8 – 1.0 mm .the female is longer and thinner ,measuring 20 -26 x 0.25 mm ,and have long uterus contains 10 -100 eggs at a time. Oviposition usually occurs in the small venules of vesicle plexus. The female, held in the gynaecophoric canal of the male,

extends its anterior end far in to smallest venules and deposits the eggs longitudinally, one at a time.

Females in copula lay eggs throughout their lives. Eggs of S*. haematopium* are oval, with a terminal spaine and production of S*. haematopium* has not been determined. The eggs pass through the genital pore located above the posterior sucker. When the worm applies the sucker to the endothelial surface, the empryonated secret lytic enzymes, enabling them to enter the surrounding connective tissue. Egg collected in the sub-mucosa before entering the lumen of bladder for *S. haematopium* (Mohmoud, 1990).

Nearly 50% of all *S.haematobium* eggs produced reach bladder and remain . Eggs must traverse the wall of the bladder before exiting with the urine.

For the life cycle to continue, eggs in urine must be deposited in fresh water. The miracidium, hatch this ciliated free-swimming seeks out its appropriate snail intermediate host so in essence, The miracidium invades the snail’s lymph spaces, and then its hepatopancreas.

A series of remarkable transformation then ensue, beginning with production of the sporocyst which in turn produce cercaria, the infectious stage for humans. Each miracidium is either male or female. cercaria accumulate at the surface of water, and swim about seeking there definitive by following gradients of chemical cues, including linoleic acid, that emanate from human skin.

Cercariae must infect within 8 hours after emerging from its snail host, otherwise they exhaust their glycogen reserves and die (Wilson, 1987).

Infection in the human host is initiated when the cercariae penetrate unbroken skin. With regards to *S. mansoni*, this step require about 0.5 hour, but occurs much more rapid with *S. japonicum* (Cheesbrought, 1998). Cercaria shed their tails, and rapidly transform within the dermal layer of skin in to schistosomula stage. After approximately 2 days, the schistosomulae migrate through blood stream to the capillaries of the lung, where they remain for another several days. It is here that the immature worm acquires their ability to incorporate host serum proteins onto their tegumental surface. This "camouflage" has the profound effect of convincing the leukocytes that the worms are "self", enabling the parasite to live out a long, prosperous life inside its new host. In addition, the worm possesses a B-2microglobulin-like molecule that aid in confusing immune defense cells, particularly macrophages, in their attempt to recognize parasite antigen. Schistosomulae migrate from the lung via blood stream to the liver, where they mature to adult worm. Both sexes produce chemo tactic agents that are mutually attractive, and eventually worm of opposite sex find each other in the vastness of the parenchymal tissue. They mate there, and migrate out into the mesenteric circulation. Egg production begins shortly thereafter. (Soulsby, 1986)

1.4 Transmission:

People became infected when cercaria-released from freshwater snails- penetrate the skin during contact with infested water. In the body, the larvae develop into adult schistosomaes. Adult worms live in the blood vessels where the females release eggs. Some of the eggs are based out of the body in the faces or urine to continue the parasite life-cycle. Others become trapped in body tissue, causing an immune reaction and progressive damage to organs (World Health Organization, 2012)

1.5 Pathology

The major pathologic lesion in schistosomiasis is the granulomatous response observed around eggs trapped in tissues (Von Lichtenberg, 1987). Each egg contains a growing miracidium, which secretes large quantities of enzymatically active and immunologically stimulation antigens as they mature. These antigens often referred to as soluble egg antigens (SEA), induce both humoral and cellular immune responses in the host (Warren, 1973). Within the egg granulomas, focal areas of necrosis are found, with deposition of eosinophilic hyaline material known as the 'Hoeppli phenomenon' (Smith and von Lichtenberg, 1967). Central necrosis and perivascular eosinophilic material decrease over time, with epitheliod cells replacing the leukocytes. Finally, a pseudotubercle is formed with foreign body giant cells surrounding the dead egg (Cheever and Powers, 1971; Elliott, 1996b).

The outcome of this granulomatous process ranges from complete healing without residua, to scarring of intestinal or vesicle walls progressing to dense deposits of collagen in the liver and bladder (von Lichtenberg, 1987). Exuberant granulomatous inflammation and adjacent tissue damage is particularly common during acute infections or reinfections. During chronic infection, most infected hosts appear to downregulate and refine granulomatous inflammation, a process termed 'immune modulation' (Boros *et al.*, 1975; Olds and Stavitsky, 1986). This results in less adjacent tissue injury but continued efficient egg destruction. This beneficial immunologic adjustment has been studied extensively in the mouse model and is immunologically mediated. Mimicking this process artificially forms the basis of several 'anti-disease' experimental vaccines (Bergquist and Colley, 1998). Despite these modulating forces, dense deposits of collagen and glycosaminoglycans can be found in many older adults chronically exposed to this parasite. This accumulation of extracellular matrix causes the major pathologic lesions observed in chronic schistosomiasis: obstruction to portal blood flow, bleeding esophageal varies and urinary obstruction.

1.6 Pathogenesis:

The terminal-spine eggs of *S. haematobium* may erode blood vessels and cause hemorrhages. Schistosome eggs, deposited in the tissues, act like foreign protein and have an irritative effect leading to round cell infiltration and connective tissue hyperplasia. The tissue reaction in these cases produces what is

known as formation of a "pseudotubercie" around each egg (egg-granuloma). The early nodules are highly cellular and are composed of eosinophils, giant cells, monocytes and lymphocytes; later on, the cellular reaction tends to disappear and is replaced by a whorl of fibrous tissue, in the centre of which degenerated and calcified eggs may be found. Large and progressive granulomas are found only around the eggs and may cause a diffuse fibrosis(Wilkins and Gilles, 1987a).

1.7 Urinary pathology:

S.haematobium affects primarily the lower urinary tract and secondarily the lungs. Adult worms of this species live in the vesicle vasculature. The eggs are laid in the mucosa and submucosa of the urinary bladder and the lower parts of the ureters. The granulomatous reaction is initially highly cellular and results in large polypoid lesions (Smith *et al*., 1974). These may cause acute obstructive uropathy. Later, the lesions become relatively a cellular and fibrotic. At this stage, lesions are called `sandy patches'. The rectum, seminal vesicles, urethra and ureters may also be involved. Without the hepatic filter present in hepatic schistosomiasis, eggs may migrate to the lungs. Ova performing the urinary tract lead to both microscopic and macroscopic hematuria and proteinuria (Wilkins and Gilles, 1987a).

The bladder lesions may calcify or deform. Sloughing and ulceration of the bladder mucosa may occur in early phase of the

disease and chronic ulceration may occur during chronic infection. Both acute inflammation and chronic scar formation can lead to unilateral or bilateral obstruction of the ureters. Chronic stasis in the urinary tract also predisposes to renal calculi and recurrent urinary infections, particularly with *Salmonella* (Young *et al.*, 1973).

Immune complex-mediated glomerulosclerosis has also been reported with all forms of schistosomiasis (Andrade and Rocha, 1979). Involvement of the mesangium with electron-dense deposits is characteristic. This complication is thought to represent deposits of immune complexes stimulated by the chronic inflammation (Sobh *et al.*, 1987).

Chronic inflammation in the urinary bladder is strongly associated with malignant transformation (Cheng and Mott, 1989). Squamous cell carcinoma of the bladder has been clearly associated with urinary schistosomiasis for many decades (Elsebai, 1977; Smith and Christie, 1986).

1.8 Immunity to schistosomiasis:

There are at least three separate and distinct aspects of the complex immunologic interaction that takes place between man and this multicellular helmint. The first is immune evasion, which allows developing parasites and adult worm to survive within the human vasculature for many years (Maizels *et al.*, 1993). The second is the complex immunological host reaction to parasite eggs, which is important for egg transport, induces most of the

clinical pathology, and is the target of host modulating responses that attempt to destroy trapped ova and yet minimize second tissue damage (Doenhoff *et al*., 1986; Hernandez *et al*., 1997a).

Finally, humans chronically infected with schistosomes appear to develop partial acquired resistance to new invasions by schistosomule (Butterworth, 1998; Capron, 1992). As a result, older humans, chronically exposed to schistosomes, appear relatively resistant to new infection (Colley *et al*., 1986; Butterworth, 1998).

1.9 Diagnosis:

1.9.1 Current approach

Since most people infected with schistosomiasis are asymptomatic, a high index of suspicion is required to clinician to identify infection, especially in geographic areas where infection is uncommon. The diagnosis should be considered in any patient with possible exposure history who presents with fever, eosinophillia, hepatosplenomegaly, anemia, hematuria, obstructive uropathy, recurrent urinary tract infection (especially with *salmonella*), glomerulonephritis seizers, transverse myelitis, pulmonary hypertension or cor pulomnale.

Hematuria is often use as a marker for infection in endemic areas and empiric treatment initiated without parasitological conformation (Taylor *et al*., 1990).

1.9.2 Urine diagnosis:

The eggs of *S. haematobium* are passed in the urine with diurnal periodicity, with peak excretion between mid-morning and mid-after noon (Doehring *et al*., 1985). Urine collected during this period may be concentrated by simple sedimentation or passing the urine through a cellulose filter to concentrate the parasite eggs. The latter allow quantification of infection. Filtration techniques that give quantitative assessment of egg excretion are replacing the simple sedimentation or centrifugation techniques (Dazo and Biles, 1974).

1.9.3 Viability test:

Viable ova can be hatched from specimens and the diagnosis made by examining for meracidia (Braun-Munzinger and Southgate, 1993). Mixing the ova in water and exposing them to light results in meracidia in the supernatant in a few hours. All species of schistosomiasis can be diagnosed using a variety of these egg-hatching techniques (Weber, 1973).

This technique is quite labor-intensive and non-quantifiable, but they can be more sensitive than a single urine examination (Braun-Munzinger and Southgate, 1993)

1.9.4 Skin test:

There is an immediate sensitivity reaction following with 15 minute of intradermal injection of antigen, usually an extract of adult schistosome's worm. A wheal and flare. The test is so sensitive or non specific (according to the quality of antigen) must

to be use less. Many people who have never left Europe give a positive reaction (probably due to contact with avian schistosomes cercariae) and many patients with long established infection are negative (Fulford *et al.*, 1991).

1.9.5 A fluorescent antibody technique (FAT):

FAT had been employed for the serological diagnosis of the schistosomiasis, using both cercaria and meracidia as antigen. It is true antigen –antibody reaction and becomes positive in early stage of infection

1.10 Epidemiology:

An estimated 85% of the world's cases of schistosomiasis are in Africa, where prevalence rates can exceed 50% in local populations*. S.heamatobium* are distributed throughout Africa; only found in area of the Middle East. Many countries endemic for schistosomiasis have established control program, but others have not. Countries where development has led to wide spread improvements in sanitation and water safety, as well as successful schistosomiasis control program, may have eliminated this disease. However, there is currently no international guideline for certification of elimination (World Health Organization, 2010).

All ages are at risk for infection with fresh water exposure in endemic area. Swimming, bathing, and wading in contaminated freshwater can result in infection. Human schistosomiasis is not acquired by contact with saltwater (ocean or sea). The

distribution of schistosomiasis focal and determined by the presence of competent snail vectors, inadequate sanitation, and infected humans. The geographic distribution of cases of schistosomiasis acquired by travellers reflects travel and immigration patterns. Most travel-associated cases of schistosomiasis are acquired in sub Saharan Africa. Sites in Africa frequency visited by travelers are common site of infection. This site include rivers and water source in the banfora region (Burkina Faso) and area populated by the Dogon people Mali; lake Malawi; lake Tanganyika; lake Victoria; the Omo River (Ethiopia); the Zambezi River; and the Nile River However, as visitors travel to more uncommon site, is important to remember that most freshwater surface water soars in Africa are potentially contaminated and can be sources of infection. A local clime that there is no schistosomiasis in a body of freshwater is not necessarily reliable(Centre for Disease Control 2014). The specific snail vectors can be difficult to identify, and infection of snails with human schistosome species most be determined in the laboratory. The types of travelers and expatriates potentially at increased risk for infection include adventure travelers, Peace Corps volunteers, missionaries, solders, and ecotourists. Outbreaks of schistosoiasis have occurred among adventure travellers on river trips in Africa. (Centre for Disease Control 2014)

1.11 Treatment

Infection with all major *Schistosoma* species can be treated with praziquantel. The timing of be treatment is important since praziquantel is most effective against the adult worm and requires the presence of mature antibody response to the parasite. For travelers, treatment should be at least 6-8 week after last exposure to potentially contaminated fresh water. One study has suggested an effect of praziquantel on *schistosoma* eggs lodged in tissues. Limited evidence of parasite resistance to praziquantel has been reported based on low cure rates in recently exposed or heavily infected populations;however, widespread clinical resistance has not occurred. Thus, praziquantel remains the drug of choice for treatment of schistosomiasis. Host immune response differences may impact individual response to treatment with praziquantel. Although a single course of treatment is usually curative, the immune response in light infected patients may be less robust, and repeat treatment may be needed after 2-4 weeks to increase effectiveness. If the pretreatment stool or urine examination was positive for schistosome eggs, follow up examination at 1 to 2 months post-treatment is suggested to help confirm successful cure. (Centre of Disease Control).

The standardadvised dosage, is a single oral dose of 40 mg per Kilogram of body weight (Kg/ bw) for all species, except for *S. Japonicum* and S. Mekongi, were the current regimes are either three doses each of 20 mg/kg bw or two doses each of mg/ kg bw, all given in one day (Von Lieshout, 1987; WHO, 2012).

1.12 Prevention and control

The global strategy for schistosomiasis is reduction of morbidity which based on treatment and vectores control. In a control program, in the firest place information should be obtained on the extent of the problem.Control program, which had been done by defferent method. By activating save construction food bridge a cross infested area and stream, recreational bathing site, especially for children. Destroying snail intermediate host, mainly by using molluscides where this is affordable and feasible and will not harm plant life. Biological control by snail eating fish or bird has little success. Treating water supply using a chlorine disinfectant were possible, storing water for 48 hours to allow time for many cercaria to die, using filter system at water inputs to prevent cercaria from entering.(Dazo and Biles, 1974).

1.13 Schistosomiasis in Sudan

Historically speaking the ancient kingdoms of the Nile basin have always been in close touch. This they did through trade, invasion, immigration due to political oppression or natural disasters such as floods, drought and famines (Archibald 1933).

The disease was will know and documented among the ancient Egyptians. It is still endemic amongst rural population. The history of schistosomiasis in other parts of the Nile valley is not clear. However, the disease is known to occur along the shores of Lake Victoria, Lake Tana and also all along the course of the Nile down

to Egyptian Delta. It is well know that the Egyptians have invaded and dominated the Nile valley during ancient times and also during the 19th century as a part of the Ottoman Empire. (Elltayeb, 1998).

The first three cases of Bilharzia were reported by (Balfour, 1903) who reported in the following year that 73 pupils of school children in khrtoum were infected with Bilharziasis.

Amongst the finest documented papers about schistosomiasis in the sudan was published by Archibald in 1914, when he reported cases of schistosomiasis associated with high pyrexia, Five years later (1918) Christopherson wrote about endemic in all provinces of the sudan except the Red Sea prpvince; and that 30% of the school children in Wadi Halfa were infected with schistosomiasis. He also reported that schistosmiasis common along the banks of the Blue and White Nile south of Khartoum (Humphery, 1932)

A considerable number of studies have been carried on *S. haematobium* infection in the Sudan. In (1933) urinary schistosomiasis was reported in Kordofan and in Darfour regions (Archibald, 1933).

Rationale:

Urinary schistosomiasis remains a major health burden in disease-endemic areas of Africa and the Middle East, affecting more than 110 million people in rural, agricultural, and periurban areas.

Individuals infected by S. haematobium frequently experience dysuria, pelvic pain, and hematuria, and are at risk of developing bladder cancer or renal failure later in life. In addition, schistosome infection is significantly associated with anemia, impaired growth, and impaired development and cognition. Consequently, schistosomiasis affects not only the health of individuals, but also the economic strength of an affected area. So this study was conduct to identify intensity of S. haematobium infection through egg count on correlation with clinical symptoms.

Objectives:

General objectives:

- To determine prevalence of urinary schistosomiasis in Helt- Ali area- Gezira State

2.2- specific objective:

- To determine the intensity of S.haematobium in infected people in study area.

- To detect haematuria, protinuria, by urine strip test

- To monitor eosinophillia in blood of infected individual

Chapter Two

Materials and Methods

Chapter Two

Materials and Methods

2.1 Study design

This is a descriptive, cross-sectional study of quantitative and qualitative variables to assess intensity of *S. haematobium* infection.

2.2 Study area and population

The study was conducted in Helt- Ali which situated in Gezira State, in about 100 km south to Khartoum, The inhabitants in this area are mainly peasant farmers who use the fertile land along the banks of the canal and its tributaries for farming.

2.3 Study variables

- Results of urine examination for *S. haematobium*

- Results of egg count per 10 ml of urine using sedimentation technique

- Frequency of haematuria

- Age measured by years

- Proteinurea

- Eosinophillia

2.4 Sample size

Two hundred urine samples were collected

2.5 Sampling

20 ml of clean-catched midstream urine samples were collected in 50 ml capacity, wide-mouth and leak proof universal containers by subjects themselves, who were previously instructed

2.6 Detection of blood in urine

The sample collection was carried out on each occasion between 09:00 am and 01:00 pm. The presence of haematuria in urine sample was observed visually and confirmed by demonstration of sedimented erythrocytes in centrifuged urine

2.7 Method of albuminuria

Albuminuria was detected by strips test. The strip was immersed in urine and the reading was based on visual aid

2.8 Method of eosinophillia

For detection of eosinophillia, blood was drawn on clear slide, air-dried, fixed with methanol and stained with Giemsa (conc.30% for 10 minute). The film was examined microscopically by oil immerse lens

2.9 Methodology

2.9.1 Urine sedimentation technique

Urine was collected into clean dry container, mixed well and transferred into 10 ml in to conical tube, centrifuged at slow to

medium speed (approx. 1500-2000 rpm) for 5 minute to sediment the egg. Using the Pasteur pipette, the supernatant fluid was discarded and the sediment was put on slide, covered with cover class and examined microscopically for *S. haematobium* eggs, Egg were counted using 10x objective, lens

2.9.2 Ethical consideration:

Ethical approval for the study was obtained from the Federal Ministry of Health and permission was provided from the local authorities of Ministry of Health and the National Schistosomiasis Control Program (NSCP) in Wd-Madni, Gezira State. Also permission was taken from Sheekh- Al hela and consent was obtained from the people after advocating them about the importance and objectives of the study.

Chapter Three

Results

Chapter Three

Results

3.1 Populations surveyed

A total number of 200 people were examined using urine sediment. Among them 144(72%) were children and 56(28%) were adults (Table 1)

Table (1): Number of children and adults surveyed for *S. haematobium*

Total No	Children	Adults
200	144 (72%)	56 (28%)

3.2 Over all Prevalence of *S. haematobium*

Out of 200 people examined 28(14%) were infected while 172(86%) did not reveals eggs in their urine (Table 2).

Table (2): The overall Prevalence of *S. haematobium* in the study area

Total number examined	Infected (%)	None infected (%)
200	28 (14%)	172 (86%)

Table (3) Rate of infection in adults and children

Population	Number examined	Number positive (%)
Children	144	19 (67.9%)
Adults	56	9 (32.1%)

3.3 Estimation of eggs count in infected people

The number of eggs counted in urine samples showed that 26 (92.9%) less than 50 egg/10 ml and 2 (7.1%) have more than 50 egg/10 ml (Table 4)

Table (4): Estimation of eggs count of *S. haematobium* in infected people

No. Examined	< 50 egg/ 10 ml	>50 egg/ 10 ml
28	26 (92.9%)	2 (7.1%)

3.4 Rate of haematuria in surveyed people

A total number of 200 people was examined for haematuria. Among them 31(15.5%) showed haematuria while 169(84.5%) did not reveal haematuria (Table 5).

Table (5): Rate of haematuria in surveyed people

Total No.	Haematuric (%)	No haematuric (%)
200	31(15.5%)	169(84.5%)

Out of 31(15.5%) haematuric people 24(85.7%) were infected and 4(14.3%) did not reveal infection (Table 6)

Table (6): Prevalence of *S. haematobium* according to presesnce of blood in their urine

Total No positive	haematuric	No haematuria
28	4 (14.3%)	24 (85.7%)

3.5 Rate of albuminuria in surveyed people

A total number of 200 people were examined for albuminuria. Among them (53%) showed no albumin in their urine while 47% showed albumin in their urine. The rates in those who showed ranged between few (15%), medium (30%) and high (2%) Table (7)

Table (7): Rate of albuminuria in surveyed people

Albumin in their urine

No albumin In their urine	few	Medium	high
53%	15%	30%	2%

Table (8): Number of albuminuria in infected people

Total positive	+	++	+++
28	9	13	6

3.5 Rate of eosinophil in surveyed people

A total number of 200 people were examined for eosinophillia in blood. Among them 30 (15%) were eosinophillic, among them 20 (67%) were infected and 10 (33%) were not infected (Table 9)

Table (9): Rate of eosinophilia in surveyed people

Eosinophilia in population	Eosinophillia in infected people	Eosinophilia in infected people
30 (15%)	20 (67%)	10 (33%)

Table (10) Number of eosinophilia in infected people

S. haematobium infection

Eosinophilia	Eosinophilia	Total number
8 (29%)	20 (71%)	28

Chapter four

Discussion

Chapter four

DISCUSION

In the present work, which exa.. ..d the rate of infection of urinary schistosomiasis in Helt-Ali area in Gezira State population were They depend on the waters from the canal and its tributaries and ponds for their domestic activities such as bathing, washing clothes, cooking etc. These activities may contribute to the transmission of schistosomiasis in the study area. The overall rate infection was found to be 14% comparison were previous studies by Abosalif (2004) reported that the percentage of *S. haematobium* in Sinar area was 43% the infection was higher in children than adults. Such high rate of urinary schistosomiasis in children may be due to the high frequency of water contact as they swim during-day, which is the time for release of cercariae consequently, they are more prone to infection. The low intensity of infection as determined by eggs count indicate that low numbers of eggs were reconed. This suggests that most of infection encountered were chronic cases. Parallel to that haematuria was also encountered in many surveyed individuals. Albuminuria was detected in infected and non infected people. In the infected rate it did not seem to correlate with the intensity of infection. The eosinophillia was higher in infected people compared to non-infected people. This is related to the fact that control of infection is largely based on eosinophils which when

stimulated generate major basic proteins that destroy the tegument of the schistosomule (Abdalla, 2014)

Chapter Five

Conclusion and Recommendation

Chapter Five

Conclusion and Recommendation

5.1 Conclusion

- This study indicated that S. Haematobium prevalence in Helt-Ali area

- The present study suggests that there was strong correlation between egg number and clinical symptoms mainly haematuria and eosinophillia, with less dependent on protinuria.

5.2 Recommendation

Children to be prohibited from water contact

Contentious diagnosis

Treatment of infected people

References

References

Abdalla, H S (2013). Lecture note, Role of Controlling Schistosoma Infection by Eosinophil

Abosalif KO (2004). Evaluation of Various Techniques Useded for the Diagnosis of Schistosomiasis

Andrade ZA, **Rocha** H (1979). Schistosomal glomerulopathy. Kidney Int **16: 23-9.**

Archibald, RG (1933). The endemiology and epidemiology of schistosomiasis in the Sudan. J. *Trop. Med. Hyg*. **36, 345-348**

Balfour, A (1903). Eosinophillia in Bilharzia and dracontiasis lancet 12th Dec. pp. **1649**

Beaver Pc, Jung RC, Cupp EW (1984). Clinical Parasitology 9th edition. Phild: *JMC press* **p.12-13**

Bergquist RN, **Colley** DG (1998). Schistosomiasis vaccines: research to development. *Parasitol Today* **14: 99-104.**

Boros DL, Pelly RP, **Warren** KS (1975). Modulation of granulomatous hypersensitivity in schistosomiasis mansoni imminol **116:1437-41**.

Braun-Munzinger RA, Southgate BA (1993). Egg viability in urinary schistsomiasis III. Repeatability and reproducibility of new method. *Trop Med Hyg* **96:179-85.**

Butterworth Ag (1998). Immunological aspects of schistosomiasis. *Br Med Bull* **54:357-68.**

Central for Disease control (2014): infection-diseases-related-to-travels Schistosomiasis **chapter-3**

Carter center: schistosomiasis **2008.7.17.** [Online]

Capron A (1992). Immunity to schistosomes. Opin Immunol *CUrr* 4: 419-26.

Cheever AW, **Powers** Kg (1971). Rate of destruction of Schistosoma mansoni eggs and adult worms in the tissues of rhesus monkeys. *Am J Trop Med Hyg* 20: **69-76.**

Cheng MG, **Mott KE** (1989). Progress in assessment of morbidity due to Schistosoma haematobium. A review of recent literature. In Progress in assessment of morbidity due to Schistosomiasis. *Trop Dis Bull* 85: **R1-45**

Cheesbrough M (1998). District Laboratory Practice in Tropical Counteries, Volume 1, U of Cambridge, *Great Britain*-**321-341**

Colley DA, **Barsoum** IS, **Dahawi** HSS et al. (1986). Immune response and immunoreguation in relation to schistosomiasis in Egypt. III Immunity and ongitudinal studies of in vitro responsiveness after treatment. 1986 Trans R Soc Trop *Medll Hyg* **80:952-7.**

Cruikshank A. Tropical diseases of South Sudan: their distribution: Tropical diseases of South Sudan: their distribution. [Online]

Davis A (1985). Schistosomiasis and related in Robeson D, epidemiology and community control of disease in warm climate country. Churchill living stone; **389-412 .2d edn**.

Dazo BC, **Biles** JE (1974). Two new field techniques for detection and counting of schistosoma haematobium eggs in urine samples.*Bull WHO* **51:399-408.**

Doenhoff MJ (1986), **Hassounah** O, Murare H et al. The schistosome egg granuloma: immunopathology in the cause of host protection or parasite survival?Trans R Soc Trop *Med Hyg* **80:503-14**

Doehring E, Rester U, **Ehrich** JHH *et al*. (1985). Circadian variation of ova excretion, proteinuria, haematuria and leucocyturia in urinary schistosomiasis. *Kidney Int* **27:667-71**.

Elliott DE (1996b). Schistosomiasis. Pathophsiology, diagnosis and treatment. Gastroenterol *Clin N Am* **25: 599-625**

Elsebai I (1977). Parasites in the etiology of cancer-bilharziasis and bladder cancer. *CA cancer J Clin* **27: 100-6.**

Eltayeb, M. (1998). Morbidity due to *Schistosoma mansoni*, PH.D. thesis, University of Khartoum. Faculty of Medicine.

Fulford AJ, **Mbuguna** AA.**Ocino** JH et al. (1991). Deference in the rate of hepatosplenomaly due to schistosoma mansoni infection between two areas in Machakos District of Kenya. Trans R Soc Trop *Med Hyg* **85: 481-8.**

Fisher A.C (1934). A study of the schistosomiasis among in the stanley village of the Belegin Congo. Trans. of Roy. of Roy. Society. trop. *Med. Hyg*. **28:227-306**.

Girges K (1934). Schistosomiasis (Bilharziasis). London: John Bale, Sone and Daneilson *ltd*.

Gendrel D. **kombila** M. beaudoin – Leblevec n, Richard-lenoble D (1994). on-typhoidal salmonellal in Ghabones children infected with schistosoma interclatium. *Clin infect Dis* **18:103-5.**

Hernandez HJ, **Wang** Y T, **Zellas** N, **Standecker** MJ (1997). Expression of class II but not class I major histocompatibility complex molecules is reguired for granuloma formation in infection with Schistosoma mansoni. *Eure J Immunol* **27:1170-6.**

Humphery, R M (1932). Vesical schistosomiasis in the Gezira irrigated areas, Sudan. Trans. R. Soc. *Trop. Med. Hyg* XXVI,(3), **241-243**.

Jordan P. **Webbe** G (1993).Epidemology. In Jordan P, Webbe G, stussock RF (ed.s). Human schistosmiasis. Walling ford: *CAB International*; **87-158.**

Katsurada F (1904). Shistosoma japonicum, a new parasite of man by which an endemic disease is caused in various areas of Japan. *Annt Zool Japan* **8: 146-60**.

Leutscher PD, Pedersen M, Raharisolo C. (2005); Increased prevalence of leukocytes and elevated cytokine levels in semen from *Schistosoma haematobium*-infected individuals. *J Infect Dis* 191 (10): **1639–47.**

Macdonald (1965) .The dynamics of helminthic infectionS with special reference to schistosome. Trans R soc Trop *Med Hyg* **59:489-506**.

Mahmoud AAF, **Abdel Wabab** MF (1990). Schistosomiasisi. In warren Ks, **Mahmoud** AAF (eds), *Tropical and Geographical Medicine*, 2nd edn. New York: McGraw Hill; **458-73**.

Maizels RM et al (1993). Immunological modulation and evation by helminth parasites in human populations. Nature **365:797-805**

Olds GR, **Stavitsky** AB (1986). Mechanisms of in vivo modulation of granulomatous inflammation in murine schistosomiasis japonica. I*nfect Immun* **52: 513-18.**

Olveda RM, **Daniel** BL, **Ramirez** BD, *et al*. (1996). Schistosoma Japonica in the Philipinines. The long-term impact of population-based chemotherapy on infection , transmission and morbidity. *J Infect Dis* **174: 163-72.**

Smith and **von lishtunbarg** FH (1967), the Hoeppli phenomenon in schistosomiasis IIhistochemistry. *Amj* **50:993-1007.**

Smith JH, **Christie** JD (1986). The pathobiology of *Schistosoma haematobium in* humans. *Hum pathol* **17: 333-45.**

Smith JH, **Kamel** IA, **ELwi** A, **Von Lichtenberg** F (1974). A quantitative Post mortem analysis of urinary schistosomiasis in Egypt. I Pathology and pathogenesis. AM J Trop *Med Hy*g **23: 1054-71.**

Sobh MA, **Mustafa** FE, **EL-Housseni** F *et al*. (1987). Schistosomal specific nephropathy leading to end-stge renal failure. *Kidney Int* **31:1006-11**.

Soulsby J (1986). Arthropods, Protozoa and Helminthes of Homesticated Animals. Tindall London.

Stelma FF, Talla I, **Polman** K *et al*. (1993). Epidemiology of schistosoma mansoni infection in a recently exposed community in Northen sengeal. Am J Trop *Med Hyg* **49 701-6.**

Taylor P, **Chandiwana** SK, **Matanhire** D (1990). Evalution of reagent strip test for hematuria in the control of schistosoma haematobium infection in school children. *Acta Tropic* **47:91-1001.**

Voge M, **Bruckner** Dg, **Bruce** JI (1978). *Schistosoma mekongi* sp. N. form man and animals compared with four geographic strains of *Schistosoma Japonicum*. J Parasitol **64: 577-84**.

Von lichtenbarg F(1987). Consequences of infection with schistosome. In rollinson D,simpson AJG (ed), the biology of schistosomiasis from genes to latrines-london: *Academic press*, **185-232.**

Warren KS (1973a), "History of Schistosomiasis" in Warren KS (ed). Schistosomiasis. *MIT press*: Cambridge, MA. **1852-1972**

Warren KS (1973b), The pathology of schistosome infections. Helminthol Abstr. **42:592-633**.

Weber M C (1973). Miracidial hatching in the diagnosis of billharziasis.Center *Afr J Med* **19:11-17.**

Wilkins A, Gilles H (1987a). Schistosomiasis hematobia. In Mahmoud AAF (ed.) Clinical Tropical Medicine and communicable Diseases. London: Baillier-Tindall: **333-48.**

Wilson RA (1987). Cercariae to liver worms: development and migration in the mammalian host. In Rollinson D, Simpson AJG (eds), *the biology of Schistosomes. From Genes to latrines.* London: Acdemic Press; **115-46.**

World health Organization, (2012). The control of schistosomiasis. WHO Tech *Rep ser* **728:1-49**.

world heath Organization, (1994). Infection of the schistosomaes IARC monographs on the evalution of carcinogenic risk to humans: **61 ;45-119.**

World Health Organization (2010): mediacentre,*fact sheets* **p115**.

Young SW, **Higashi** A, **Kamel** R *et al*. (1973). Interaction of salmonella and schistosomes in host- parasite relations. Trans R Soc Trop *Med Hyg* **67: 797**.