Review Article

Schistosomiasis: epidemiology and burden of disease in the Sudan

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Abstract

Schistosomiasis is an old disease known from the old time of the Egyptian Pharaos, 1500 BC. It is a parasitic disease caused by S. mansoni and S. haematobium causing a chronic inflammatory process affecting the intestine and urinary bladder leading to oesophageal varices and carcinoma of the bladder. The schistosomiasis was introduced as early as 1909 in Dongola, Sudan and Southeastern, Sudan, but reached a large scale infectivity in the Gezira population after the establishment of the Gezira cotton irrigation scheme in 1925. The Blue Nile Health Project (BNHP) was established in 1980 to control water bone disease brought the high prevalence of pre-control of 50% to 6.1% by the end of 1989. Among the milestone of the history of schistosomiasis is the discovery of antimony treatment in 1912 by the British physician Dr JB Christopherson in Khartoum Civil Hospital. Emphasis should be laid on distribution of the effective drug praziquantel to the spreading schistosomiasis in the Sudan. Lessons should be learned from the BNHP and consolidate the success of this project.

Schistosomiasis is the generic name given to diseases caused by the parasitic blood flukes of the genus schistosoma. An older name, still widely used is bilharziasis. The three major species that commonly infect man are: S. mansoni which cause intestinal disease, S. haematobium, urinary schistosomiasis, and S. japonicum a more severe form of intestinal disease and is found only in South East Asia. Schistosomiasis, an old human disease, remains one of the most prevalent infections and has significant economic and public health consequences. Despite the availability of a potent drug, praziquantel, that lead to cure and marked reduction of egg–faecal load, the control of the disease as a public health

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Schistosomiasis in the Sudan: A Review Article

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Schistosomiasis remains a challenge. Schistosomiasis leads to considerable morbidity and mortality. Among the world's serious parasitic diseases, schistosomiasis ranks second only to malaria in the number of people infected and the extent of the areas where the disease is endemic.

Although exact figures are hard to obtain, it is estimated that, out of 779 million people at risk of schistosome infection world wide, 200 million are infected, of whom three-quarters live in Africa and 20 million suffer from severe sequelae (1).

In the Sudan it is estimated that, 7 million people are infected, distributed all over the Sudan including areas like Khartoum state, not known previously to be endemic.

**Keywords:** antimonials, schistosomes, bilharziasis, Blue Nile Health Project (BNHP)

**The parasite and the host:**

Schistosomiasis is a water borne infection and its life-cycle is dependent on the presence of the intermediate host, the aquatic snails Biomphalaria for S. mansoni and Bulinus for S. haematobium. S. japonicum is viewed as a zoonotic infection as the buffalo maintain the infection in regions of Southeast Asian countries.

**Pathogenesis and pathology:**

The adult worms which live in the mesenteric veins (S. mansoni, S. japonicum) or vesical plexus (S. haematobium) provoke little inflammation in the veins. Untreated individuals can harbour live schistosomes for as long as 3-7 years.

The pathology is caused by the deposition of schistosome eggs in human tissue. The clinical manifestations of schistosomiasis reflect the immunologic and inflammatory reactions to schistosomal eggs. The basic lesion is a circumscribed granuloma or a cellular infiltrate of eosinophils and neutrophils around an egg.

Epidemiological data from endemic areas show that schistosomiasis is a disease of children of primary school age (7-16 years).

Although the disease can cause clinical symptoms calling for medical care, many patients remain asymptomatic and present only with complications (liver disease (S. mansoni) and urogenital disease (S. haematobium)).

The disease should be viewed as a chronic inflammatory disorder and a history of past infection may not be obtained. Of importance is the accumulation of childhood infection that lead to permanent disability. In a study in Gezira, Sudan, Homeida et al(2) showed that while peak prevalence in intensity of infection occurs in the teenage children, periportal fibrosis occurs and peaks 10-15 years later. Hayashi et al followed 70 Japanese schistosomiasis patients from the time of cure (S. japonicum infection) in 1966 until 1997 when only 40 of the subjects were still alive. None suffered from liver disease in 1966. Reassessment of the clinical status in 1997 demonstrated liver fibrosis in 25 cases. Abnormal liver tests which could not be associated with HBV nor HCV infection were found in 40% of the cases. Two patients developed cerebral schistosomiasis (3). It is concluded that pathology may progress and complications develop even in the absence of an overt infection or apparent cure.

**History of schistosomiasis in the Sudan:**

Schistosomiasis is an old disease. Its history dates back to the Pharaonic kingdoms 1500 B.C. Schistosome eggs have been identified in the Pharaonic mummies (4). The Asian schistosomiasis (S. japonicum) also is an old disease and eggs of japonicum infection were found in the rectum and liver in a female corpse which dates back to 206 BC.

In 1852, Theodore Bilharz a German physician identified the adult worm in his postmortem study in Egypt (5), while the life-cycle was unraveled by the Scottish parasitologist, Robert Thomson Leiper in 1912 (6,7).

In the early 1900, Sudanese patients with schistosomiasis were thought to be infected in...
Egyp. However, during the investigation of outbreak of cases of Kala-azar in eastern and south eastern Sudan in 1909 in what was called ‘The Kala-azar Commission’ the investigators stumbled on many cases of hitherto unknown ‘bilharziosis’ in the region of Singa in the Blue Nile Province. This finding was mentioned in the part of the report of the commission published by Dr Douglas Stokes Brownlie Thomson in the 4th Welcome Report published in 1911. That was the first report of the presence of the disease in the Sudan. In 1921 pumping stations were introduced to the Dongola province. Canals from these irrigated land were found to be infected with schistosomiasis, the source came from immigrants from Egypt. However, in 1925 when Sennar dam was completed and the Gezira canals were man dug with Egyptian--fellaheen (10,000 Egyptians were involved) infestation of the Gezira with schistosomiasis made this area the highest infected area.

Sudan also figured high in the history of schistosomiasis following the Christopherson's discovery, the development of tartar emetic as a cure for schistosomiasis. It was the most significant contribution to medicine at the time. John Brian Christopherson, was born in Yorkshire, U.K., and qualified in medicine from Cambridge and St. Bartholomew's Hospital in 1893 (Fig 1).

He first came to Sudan in June 1902 after a brief service in South Africa. He became the first Director of the civilian Sudan Medical department in 1904. He was also the first Director of Khartoum Civil Hospital (established 1909(Fig 2). The hospital now is the headquarters of the Sudan Medical Specialization Board (Fig 3).
He used tartar emetic for treating patients with leishmaniasis and noted the disappearance of bilharzia eggs from the urine of those with the disease.

Tartar emetic gave striking results according to Christopherson in 1918\(^{(10)}\). He reported the outcome of this chemotherapy in 13 patients with relief of symptom and disappearance of eggs from the urine. He described the toxicities of the drug and raised precautions against unsupervised use of antimonials.

Antimonial compounds in one form or another dominated the field of chemotherapy of schistosomiasis up to the seventies when more safe drugs were introduced.

The Blue Nile Health Project (BNHP) a story of success in the control of schistosomiasis:
The Gezira-Managil scheme is one of the largest irrigated schemes in the world which covers over 8000 square kilometer (over 2 million acres). Prevalence of schistosomiasis was reported to be 18% in 1950\(^{(11)}\) reaching 50% in early 1970s\(^{(12)}\). In the mid-1970s the Gezira suffered from a serious malaria epidemic. The thought of establishing an integrated water-borne disease control (schistosomiasis, malaria and diarrheal diseases) came from Mutamad Amin who presented a concept paper to the WHO (personal communication). The project was initiated in 1979 for a period of 10 years at an estimated cost of $154 million (1978 prices).

It was established with a strong Sudanese leadership and support from a variety of countries, agencies and foundations\(^{(13)}\).

The control was based on and supported all along with scientific evidence and operation research. The project was under the leadership of Ahmed Ayoub El Gadal (now professor of community medicine, Sennar University, Sudan), and strong support from Mutamad Amin, Mohamed Kardaman, Akoud, Asim Dafalla, Harivi and the British biologist Alan Fenwick (now Professor at Imperial College London).

The project was based on strong health education and community participation (village health committees were established).

The strategy to control schistosomiasis was based on supply of clean water through sand-filteration, and assistance to the population to dig pit-latrins. Praziquantel which was then newly introduced for the treatment of schistosomiasis was tested and proved suitable for mass chemotherapy. Malaria control was based on house spraying, and prompt diagnosis and treatment.

The BNHP was successful and gave an example of an integrated approach to control water borne diseases with strong community participation and community ownership. Infrastructure for domestic water-supply and latrines slabs were supplied to the villages. Diagnostic facilities and treatments were stocked and provided in all health centers.
Capacities of the qualified personnel were secured. Achievements included the reduction of the prevalence of schistosomiasis from 50% in 1981 to 6.1% by 1989. Malaria prevalence was kept below the project goal of 2% (Fig 4).

The government of Sudan and the Ministry of Health Gezira state failed to sustain the project. Recent studies have shown an uprise in the prevalence of schistosomiasis in the farmer population who live near the canals (the cambos).

In his thesis (unpublished) ElHaj M, 2009 revealed a low prevalence rate in school children from the BNHP original core villages. The overall prevalence of S. mansoni infection in 5 villages dropped from a mean of 50% to 6.1% by the end of the life period of the BNHP. However this drop continued to reach a level of 2.2% in 2009 reflecting the general improvement in the environment in these villages. The overall prevalence rate of S. haematobium was also less than 1% in these villages. Gezira villages have been by and large modernized with artisan well for water supply, electricity and schools. Most of the inhabitants of these villages abandoned the agricultural life and left the job to the immigrants from the west of Sudan, Fulani, Bergo and others who live near the canals (cambos). The overall prevalence of schistosomiasis in these cambos is between 30-40% with a shift from S-mansoni to S. haematobium. Children under 7, previously neglected and not included in most prevalence surveys and excluded from community based praziquantel distribution are now shown to be also infected. Abu-Sinn showed a prevalence of S. mansoni of 3.7% and 3.1% in 8 Combo villages (stable residency) with 8.2% in the cambos with much higher prevalence of clinical symptoms indicative of intestinal disease.

There are no structured, sustained efforts at the present time to control schistosomiasis in the Sudan. Distribution of praziquantel is limited to some schools in Khartoum and the coverage rate is low. It is not surprising that 17.5 million of the Sudan populations are at risk of the disease and around 5.5 million are infected with one of two forms of schistosomiasis with a shift in most endemic areas from S. mansoni infection to S. haematobium.
References