Schistosomiasis as a disease and its prevalence in Sudan: An overview

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1. Introduction

There are a number of digenean parasitic trematodes that are capable of causing diseases in humans. Among these, schistosomiasis (bilharzia) is the most important, and is caused by members of the genus Schistosoma in the family Schistosomatidae[1,2]. The most important schistosome species that infect man are Schistosoma mansoni (S. mansoni), Schistosoma haematobium (S. haematobium) and Schistosoma japonicum (S. japonicum); and the numbers of schistosomiasis cases are widely on the increase[3-5]. The disease transmission is usually associated with poor socioeconomic conditions[6,7]. Schistosomiasis is focally distributed with the transmission influenced by several factors. Among these factors, the distribution of the intermediate host snails is of great importance[8-12],

2. Global schistosomiasis

Schistosomiasis is the second most prevalent parasitic disease after malaria in terms of socioeconomic and public health importance in the developing countries, and it can persist for 30 years if left untreated[13-17]. Schistosome eggs have been recovered even from Egyptian and Chinese mummies that were several thousand years old[18,19]. However, scientific studies of the disease did not start until the middle of the 19th century with several independent reports. First Japanese workers described the disease as acute katayama syndrome[20], and found distome trematode in the urinogenital blood vessels during post mortem examinations of Egyptian corpses. Of the five species of Schistosoma affecting man, S. haematobium[21] are the most important and wide-spread, while the other two, Schistosoma intercalatum (S. intercalatum)[22] and Schistosoma mekongi[23] have more localized distributions[24]. Later another species, Schistosoma malayensis, was reported to infect aboriginal people in a small jungle focus in Malaysia[25].

Based on the latest extrapolations, it is estimated that 800 million people live at risk of the infection[26] and 252 million people are actually infected with schistosomiasis, of the infected people, 90% are concentrated in the sub-Saharan Africa[14]. Schistosomiasis is still endemic, but is at very low levels, in North Africa and in several countries of the Middle East. In Asia, schistosomiasis is also endemic along parts of the Yangtze River basin in China, and in the middle reaches of the Mekong River in Laos and Cambodia. It is also endemic in several eastern states of Brazil, and has several foci in Venezuela[27-29]. The main reasons for the global prevalence and increase of schistosomiasis are: (1) ever-increasing
number of irrigation systems for agriculture and cattle breeding; (2) construction of dams and man-made lakes for hydroelectric power production; and (3) civil strife and war which contribute to massive human migration[30,31]. Despite the tremendous global burden of schistosomiasis, and the likelihood of the disease gaining importance, public health significance of this disease is often underestimated. There are two common explanations for this: (1) focal distribution of schistosomiasis; and (2) the severe disease state only follows after many years of mildly asymptomatic infections [13,32-34].

3. Schistosomes life cycle

Unlike other digenea, adult schistosomes are dioecious, having separate males and females located in the mesenteric veins[8]. Essentially all schistosomes of medical importance follow the same life cycle[35]. Worms pair as adults in the liver and remain there for 3–6 years or more[36]. An adult female of schistosome discharges 20–3 500 eggs per day[8,37,38]. It is assumed that approximately 50% of the eggs pass through the colon, on the wall of the bladder or the genitourinary apparatus, and are excreted with faeces or urine. The remaining 50% of the eggs are trapped within the tissues of these organs[39]. The eggs which measure 89–140 × 60–67µm are not opeculate, and have distinctive shape and spine[40]. The eggs of these parasites excreted with urine or faeces hatch when they come in contact with freshwater under favorable conditions of temperature (25–30 °C), light, and osmotic pressure. Free swimming miracidia emerge from the egg shells and can remain infective for 8–12 h. The miracidium penetrates the specific cold-blooded intermediate host snails in the water, predominantly via the foot of the snail[37,41,42]. After penetration into the snail, the miracidium sheds its epithelium and begins development into a mother sporocyst, usually near its point of entrance, and within a period of 4 weeks the mother sporocysts give rise to daughter sporocysts which differentiate into cercariae by asexual multiplication. The cercarial output from an infected snail has been estimated to be about 1 500/day for both S. haematobium and S. mansoni, and this lasts for up to about 18 days. The period from the penetration into snail to the release of mature cercariae is about 4 weeks as reported by Webbe[43], however, for S. japonicum it takes approximately 8 weeks for the development of a miracidium into a cercaria at water temperature of 24–26 °C[37]. The cercariae are released on a circadian (24 h) cycle during daylight hours into water, and swim freely searching for a suitable vertebrate host within 24–48 h[44]. When definitive hosts, humans or other warm-blooded animals, come in contact with the infected water bodies, the cercariae can bore through the skin of the host with the assistance of lytic substance from the penetration glands[37,45,46]. The tails of the cercariae fall off and the bodies transform into schistosomula under the skin. Within 48 h, the schistosomula enter the peripheral blood vascular system to be carried passively to the lungs via the right side of the heart, then via the pulmonary veins to the left side of the heart. From the heart, the schistosomula are carried to the systemic circulation where an individual schistosomulum may take several circuits of the pulmonary systematic circulation before entering a blood vessel which leads to the hepatic portal system in the hepatic portal vessels. Period of growth and maturation then follows before the adult worms migrate either to the superior or inferior mesenteric veins (S. mansoni, S. japonicum, S. intercalatum) or the vesical plexus (S. haematobium) [45].

4. Schistosomiasis pathology

Different kinds of health complications associated with schistosomiasis may appear according to the parasite species, and or to the human population characteristics[24,33,47]. Serious and very common complications of the disease include bladder or urotre calciﬁcation in urinary schistosomiasis, and enlarged liver and spleen in intestinal schistosomiasis[48]. Also periporal fibrosis of the liver characterizes S. japonicum infection, in which the eggs frequently calcify in tissue; but portal and pulmonary hypertension are potential complications of S. mansoni infection[49]. In addition, in the Far East, infection with S. japonicum known as ‘Katayama’ disease is characterized by growth retardation in children, acute cerebral manifestations, enlargement of the liver and spleen, bloody diarrhea, and occasionally fever (the so called ‘Katayama fever’)[37,50,52].

5. Schistosomiasis intermediate-hosts

Schistosomes are transmitted by freshwater snails of the class Gastropoda in the phylum Mollusca. Many subspecies of the amphibious snail, Oncomelania hupensis, transmit S. japonicum, and two species of the truly aquatic Basommatophora genera, Biomphalaria and Bulinus, snails, transmit S. mansoni and S. haematobium, respectively. While Bulinus forskalii snail transmits S. intercalatum and Bobertia kaporensis transmits Schistosoma mekongi[13,29,53-55].

6. Schistosomiasis control

The control strategies of schistosomiasis are based on health education, selective but large scale chemotherapy, improvement of water supplies and sanitation, and control of the intermediate host snail[13]. Health education aims to promote and reinforces healthy behavior with full participation of both an individual and the community. Changing faulty human behaviors could largely prevent schistosomiasis, and health education is of paramount importance to achieve this[13,44]. World Health Organization reported that praziquante is an active antischistosomal drug, and since its discovery the disease treatment strategies have changed[56,57]. Nevertheless, without an integrated control program, re-infection can quickly follow and treatment must be sustained[58]. Snail control is most important to the control schistosomiasis transmission, and may include the use of chemical molluscicides, biological control agents and environmental management[13,59-62]. Past efforts to control snail populations through the use of chemical molluscicides or alteration of their habitats have resulted in environmental pollution and damage[63]. Environmental management includes drainage, filling in, and the lining of canals with concrete, etc., but these methods are generally expensive though long-lasting. Application of the genetic manipulation in a certain population of snails should also be in consideration[64].

7. Schistosomiasis in Sudan

The disease has existed in Sudan as far as back as 2600 B.C, and became endemic and a major public health problem in most sudanese states as water resources development projects expanded[65]. It is also due to political and economic contacts with Egypt, and to the thousands pilgrims who flow through the country on their travel between Mecca and the West African countries where the disease is
known to be endemic. In recent years, immigration and the increase in travel from high-risk areas have led to an increase in the number of imported cases[96].

The first case of the disease in Sudan was reported by Balfour, who found 17% of the children in Khartoum primary school suffering from urinary schistosomiasis[67]. In 1919, Egyptian labourers were employed to dig a canal for a pump irrigation project at Nuri in the Northern State, and the disease spread to other irrigation farms and became widespread in the Dongola area[65]. At the same time, several foci of schistosomiasis were discovered in Kordofan area, at Um Riwaha, and Abu Zabad. By the year 1925, a new era began in Sudan with the opening of an agricultural scheme in the Gezira State. It was estimated that the overall prevalence of the disease in Sudan in 1949 was 8.1% for S. haematobium and 5.4% for S. mansoni[68]. At Shambat, North of Khartoum, the capital, the prevalence of S. haematobium in 1959 was 22% among school children[69]. In 1972, a study was conducted at the Gorashy Dairy Farm, Khartoum, showing an infection rate of 65%, and of 37% among employees and labourers not working in the field[70]. It was reported that prevalence of S. haematobium ranges from 15% to 70% in school children nearby the village of Koher, in Khartoum[71]. While the prevalences 48% and 37% for S. haematobium and S. mansoni, respectively, was reported in El-Sileit area of Khartoum. However, S. mansoni was found at the reasonably high 23% prevalence in the Gommoeya Scheme of Omdurman area[72]. Some years later, for the same scheme the prevalence rate was reported to be 33% of S. mansoni[73]. The East Nile Locality, which is characterized by extensive cultivated belts, is presumably the only locality harbouring both these infections with a wide distribution[74]. Recent epidemiological surveys that elucidated the prevalence and intensity of these infections have presented alarming figures[44,75].

Bella et al. reported an overall prevalence of 57% S. mansoni[76], while Fenwick found overall prevalences of 67% and 17.9% for S. mansoni and S. haematobium, respectively, in villages of the Gezira scheme[77]. Omer et al. found that the prevalence of S. haematobium among fishermen in Lake Nasser was over 30%[78]. In Southern Sudan both S. mansoni and S. haematobium were reported[79]. While assessing the possible ecological effects of the Gounglei canal, Southern Sudan, the presence of schistosome intermediate hosts was observed in the area, and the possibility was raised that the canal might increase the snail populations and the prevalence of parasitic diseases. In Western Sudan, S. haematobium prevalence of 14% was reported among children[80], but later the prevalence of S. haematobium was found to be 8.5%, 29.5%, 35.8% and 32.7% among children in Kadogli, Deleng, Rahad and Um Rawaha, respectively[81]. Few foci of S. mansoni transmission in Darfur Mountains have been recorded[82], but later it was reported that infection with intestinal schistosomiasis is quite prevalent among villagers and school boys in Jebel Marra, where prevalence and intensity of infection reached to 24.7% and 84.8 epg, respectively[83]. In Eastern Sudan, earlier reports suggested that in Kassala State, S. haematobium occurred in association with marshes and rain water pools[84]. However, hospital reports showed the prevalence of S. haematobium at 8.7% in Kassala and at 3.4% in Gadaref[85], while in Khartoum both types of schistosomes have been reported[86]. Their prevalences increased after extension in irrigated areas around Khartoum[87]. Because of the expansion in water resource projects, schistosomiasis increased and became endemic in these projects, in New Halfa Scheme[75], in Rahad Scheme[80,91], and in Gumaid Sugar Cane Scheme[44,92]. In the Managil extension area, a study showed that the prevalence of S. mansoni infection ranged between 33.3% and 54.8%(93). Surveys in Kenana Sugar Cane Scheme, in the White Nile State, revealed prevalences of S. mansoni and S. haematobium at 24% and 12%, respectively, after a few years of operation[94]. Meanwhile, the overall prevalence of S. haematobium in Um Hani scheme, South of Kosti in the White Nile State, was 53.8%(95), and the overall prevalences of infections by S. mansoni, S. haematobium or both species were 10.1%, 21.4% and 4.5%, respectively, among 936122 children from 27 schools in the former White Nile State[96].

Nowadays, schistosomiasis is prevalent in all states of Sudan, and it has increased in distribution and prevalence as a result of the progressive expansion in water resource development, and with increased population movement[97,98].

Conflict of interest statement

We declare that we have no conflict of interest.

References


[23] Voge M, Bruck D, Bruce JJ. *Schistosoma mekongi* species from man and animals compared with four geographical strains of *S. japonicum*. *J Parasitol* 1978; 64: 484-577.


[61] Sulieeman YA. Laboratory and field investigations on population dynamics and biological control of Oncomelania hupensis snail, the intermediate host of Schistosoma japonicum [dissertation]. Xianmen: School of Life Sciences, Xianmen University; 2013.


