

Schistosomiasis. Experience with Transfer Factors
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Schistosomiasis is a widespread parasitic disease, which affects about 200 in seventy epidemic collections of countries of the world. million people and at risk of infection 500 million people.

Nosological forms: genitourinary, Japanese intestinal, intercalate, and Mekong.

Economic damage: Only at the expense decrease in working capacity the annual loss from full or partial disability is: US \$ 500 million in Africa; US \$ 20 million in Southwest Asia; US \$ 120 million in Southeast Asia; US \$ 70 million in America. The total annual damage worldwide is estimated at US \$ 897,790,450 (excluding the cost of maintaining health facilities and sick pay).

Historical excursion: The center of origin of molluscs and schistosomes was the region of the African Great Lakes in the East African Highlands, the Nile basin, Egypt.

The discovery of the paleontologist Ruffer (1910) in microscopic sections of the kidneys of Egyptian mummies of the XX dynasty (1200-1130 BC) of a large number of calcified eggs of the causative agent of urogenital schistosomiasis - *Schistosoma haematobium*. The papyri indicate the epidemic nature of the disease, in the "Book of the Dead" the connection between the disease and water is noted and examples of punishment for its pollution are given. Books by Arab historians of medieval Egypt, in which it was noted that among the men of Egypt, the disease is widespread, from which they "menstruate like women." Due to the excretion of blood in the urine - the main symptom of genitourinary schistosomiasis - the disease is called "Egyptian hematuria". In the 19th century, doctors Renault, Larrey, Pruner, participating in the Egyptian company of Napoleon in 1799-1801, noted the significant spread and severity of the course of Egyptian hematuria among the local population,

The most intense focus arose in the interfluvium of the Tigris and Euphrates, where a system of irrigation canals irrigated the land around the clock.

In Southeast Asia (Yangtze basin), Japanese schistosomiasis in China for more than 2 thousand years is evidenced by the discovery of schistosome eggs in the tissues of a mummified corpse of a woman (Tsao Hs-ro-Ting, 1975).

Brought to the American continent in large quantities the importation of slaves from Africa blacks in the XVI-XVIII centuries.

In subsequent centuries goes on accumulation data O morbidity, clinic.

In 1904, Katsurada discovered *S. japonicum* in Japan. Gonzales-Martinez identified her in Puerto Rico; Catto - in Singapore; Hogan is in China. In 1904-1925. schistosomiasis found in the Philippines, Brazil, countries. Venezuela and others

Russian researcher S.S. Abramov in 1906 G. described pathological picture of urethritis and cystitis caused by genitourinary schistosomiasis.

Fujinami and Nakamura in 1909 proved that the gateway to *S.*

japonicum are the skin, and natural hosts are humans, cattle, horses, dogs, and cats.

The intermediate host is mollusks of the Hydrobiidae family (Miyar and Suzuki 1913–1914, Leiper established in 1915 *S. haematobium* and *S. mansoni* are widespread in Egypt - the intermediate host of the mollusks *Bulinus contortus* and *Planorbis boissyii*. 1924 Faust and Melency deciphered the vital cycle of *S. japonicum* and pathomorphological changes in the organs of the final host.

In 1918, Christophersen proposed to treat schistosomiasis with antimony preparations, Chandler proved in 1920 the effectiveness of copper sulfate in the fight against molluscs. In 1928, Schmiolt developed a method for treating schistosomiasis with stibofen. In Japan, the fight against shellfish with calcium cyanide.

Etiology. Distributed: Africa, Asia, Latin America. Common names for this group of diseases are: Schistosomatoses (lat.), Stosomcasis, bilharziasis (English), *S. bilharzioses* (French), Bilharziakrankheit (German), esquistossomose (Spanish).

In humans, 9 types of pathogen parasitize:

1. Genitourinary schistosomiasis (*S. haematobia*);
2. Intestinal (*S. mansoni*);
3. Japanese (*S. japonica*);
4. Intercalate schistosomiasis (*S. intestinalis intercalatum*);
5. Mekong schistosomiasis (*S. Mekong*); less often,
6. *S. bovis*;
7. *S. mattheei*;
8. *S. margrebowiei*;
9. *S. rodhaini*;

Schistosomatoid dermatitis is caused by: *S. spindale*, *S. douthitti*, *Heterobilharzia americana*, *Trichobilharzia ocellata*, *T. Stagni cola* and others.

Veterinary medicine: *S. bovis*, *S. mattheei*, *S. nasalis*, *S. spinolale*, *S. indicum* and *Orientobilharzia turkestanicum*.

There is evidence that hybridization is possible between *S. haematobium* and *S. intercalatum*; *S. haematobium* and *S. matthei* in the final host.

S. Haematobium is the causative agent of genitourinary schistosomiasis.

Adult helminths parasitize in the veins of the urinary bladder, mesentery and portal vein in humans and monkeys.

Male: 10-15 mm long and 0.75-1 mm wide, grayish-white, cuticle with small papillae, at the front end of the oral and abdominal suckers. Near the abdominal sucker, there are 4–5 testes connected to the genital opening. On the ventral side of the male, there is a longitudinal groove in which the female is placed.

The female is 20–26 mm long, 0.25 mm wide, the color is darker due to black and brown inclusions of blood pigment in the intestinal wall. It has oral and abdominal suction cups; the ovaries are located in the posterior half. In the uterus, 10–100 eggs can develop simultaneously.

Oviposition occurs in small terminal venules of the bladder, less often in the rectum and mesentery. When the bladder contracts and

rectal eggs penetrate into their lumen. Color yellowish-brown, transparent shell with a thorn at the pole of the egg, sizes 0.12-0.16 0.4-0.6 mm. The productivity of the female is 20–300 eggs per day.

Life cycle: egg miracidium stage I sporocyst (maternal)
stage II sporocyst (daughter) cercarium schistosomula adult
parasite.

Infects human cercariae through the skin schistosomula migration along the lymphatic and circulatory pathways through the heart and lungs to the liver, where the parasite matures. The time from the injection site to the liver is 10-15 days. Further migration to the mesenteric veins, veins of the bladder, where egg laying takes place. The period from implantation to oviposition and their appearance in the urine or feces of the final host is on average 30-45 days. The life span of the parasite in the human body is 5–10 years (up to 28 years).

Epidemiology

Infection occurs through contact with the skin and mucous membranes of living cercarias, in contact with water.

Japanese schistosomiasis is also infected by contact with soil and vegetation on the shores of water bodies and pastures (mollusk *Oncomelania*).

The source of the invasion of *S. haematobium*, *S. mansoni*, *S. intercalatum* is a sick person who excretes eggs with feces. Along with humans, the source of *S. mekongi* invasion is the dog, while in *S. japonicum*, in addition, domestic and wild animals (cattle, small cattle, horses, pigs, dogs, cats, rodents). A source of invasion in 30–45 days.

Pathogenesis:

1. Sensitizing effect of metabolic and decay products
helminths and their eggs.
2. Their toxic effects.
3. Mechanical damage organs and fabrics cercariae, schistosomules, sexually mature parasites and their eggs during migration.
4. Toxic effects on the body of its own decay products
body tissues.

Disease stages:

I. The stage of implementation (2–4 weeks): on the first day, mechanical and chemical damage to the skin and mucous membranes by cercariae. In 8 minutes it is able to penetrate the epidermis, in 20 minutes - to reach the lymphatic vessel. It is accompanied by vasodilation of the surface layer of the skin, infiltration with eosinophils and neutrophils, itching and edema, petechial-papular rash.

II. The stage of migration of schistosomules from the site of introduction through the right departments the heart to the lungs and the portal vein system. It is accompanied by focal lesions of the pulmonary capillaries, manifested by edema, hemorrhages of the lung parenchyma, inflammation of the bronchioles, catarrhal pneumonia, rarely - pulmonary vascular thrombosis.

III. Maturation stage of schistosomes: occurs mainly in the veins liver, less often the spleen and intestines, which leads to some increase in these organs. Inflammation of the central nervous system with *S. japonicum* lesion. Maturation of an individual is concentrated in the portal vein in a countercurrent movement into the mesenteric veins (*S. mansoni*; *S. japonicum*; *S. intercalatum*), into the veins of the genitourinary organs (*S. haematobium*).

The metabolic products of worms and the laid eggs cause a second wave of sensitization.

Case from practice

Patient K. turned to the Eliseeva Center with complaints: frequent loose stools with bloody discharge for 15 years. Clinical diagnosis: ulcerative colitis. For several years he took Salafalk - which gave an unstable remission, later caused an allergic reaction.

Objectively: the patient is malnourished, anemic, the skin is earthy, the turgor is reduced.

Blood test: Hb - 78 units; E - 2.5.

Irigoscopy (Fig. 1, 2).



Rice. one.S. mansoni: Multiple filling defects with contrast masses in the descending, colon and sigmoid colon.



Rice. 2.S. mansoni: Significant defect in the filling of the contrast material sigmoid colon.

MP testing by the ART method: BI, FI, immunity: exhaustion, bacteria, viruses; worms: Mansonia schistosoma ↓ + Large intestine ↑.

I carry out therapy:

1. BRT endogenous No. 10.

2. EPT: frequencies: colitis, ulcerative colitis, ulcers, viruses, bacteria, schistosoma - No. 10.

3. Transfer factor according to the scheme developed by us.

After 10 days: stool without blood. Normal leather. Blood test: Hb - 140, E - 4.2. Colonoscopy 1 month later: somewhat incomplete

epithelialized erosion.

The patient continues to be monitored.

Conclusions: complex treatment with BRT, EPT and transfer factors contributes to significant improvement in the treatment of severe chronic pathology.

Literature

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E.V. Tikhonov Schistosomiasis. Experience of using transfer factors // XII