



I'm not robot



Continue

Schistosoma species pdf

Human infection with schistosoma worms causes significant morbidity and mortality worldwide (15). Five clinically important species cause the bulk of human infections. These are Schistosoma Hematobia, S. Mansoni, S. Japonicum, S. Intercalatum and S. Mekongi. Humans can also be infected with animals or avian schistosomiasis, but this leads to no or little morbidity. Adult schistosomes parasitize specific locations of the venous system: pelvic plexus (S. haematobium) and mesenteric veins (other species). After maturation, female worms lay their ova intravascularly. Some of the eggs run from lumen veins to nearby viche, which will be excreted with urine or feces. Schistosome hatch eggs upon reaching fresh water in the environment releasing miracidia, which search and infect specific intermediate snail host. The next stage, cercariae, is released from infected snails; they are capable of penetrating unexposed human skin. Upon entering the dermis, cercariae change in the stage of schistosomula and migrate to the host tissues through the lungs and liver to reach their final habitat. The epidemiology of Schistosoma hematobia is found in Africa and the Middle East; S. mansoni is found in Africa and Brazil; S. japonicum in China and the Philippines; S. intercalatum in West and Central Africa; and S. mekongi in Southeast Asia. In the United States, although schistosomiasis occurs in immigrants from endemic countries, transmission does not occur because there is no appropriate intermediate host of the snail and there is sanitary removal of feces and urine (biofluids, which contain derived eggs of schistosoma). Clinical manifestations of three different clinical conditions occur in schistosomiasis and are associated with certain stages of the parasite. Cercarial dermatitis occurs after exposure. The papular itchy rash, also called itchy swimmers, is more noticeable in infection with avian species. Acute schistosomiasis or Katayama fever coincides with the maturation of adult worms and the onset of oociposition. It is a serum-like syndrome and occurs in severe infections of infectious-naive people, especially with S. japonicum. Patients are feverish with flu-like symptoms including cough and headache. They often develop hepatosplenomegaly, lymphadenopathy and outstanding peripheral eosinophilia. Chronic schistosomiasis occurs due to egg retention in the host tissues, resulting in the formation of granulomas. Infections of S. japonicum and S. mansoni lead to chronic intestinal and liver dysfunction. Fatigue, colic abdominal pain, diarrhea, and anemia due to blood loss from intestinal ulcers occur with intestinal schistosomiasis. Eggs in the circulation portal can cause presynusoidal blockage of portal blood vessels, leading to portal hypertension. Chronic schistosomiasis often presents with hepatosplenomegaly and can progress to decompensated liver disease. Eggs can also penetrate the lungs and granulomatous pulmonary blood vessels, which leads to pulmonary cortex. S. hematobia infections involve urethra and bladder and other pelvic organs. These patients are present first with hematuria and dysuria, and then with obstructive uropathy and urmia. Flat cell bladder cancer is known to occur in patients with chronic S. haematobium infections. Participation of the central nervous system in schistosomiasis occurs infrequently. S. haematobium and S. mansoni most often affect the spinal cord, while S. japonicum affects the brain. Laboratory diagnosis of human infection by any type of schistosomiasis is based on obtaining an accurate geographical history and experience that can lead to exposure. A thorough clinical and laboratory assessment is mandatory. The final diagnosis depends on the demonstration of schistosomal eggs in the urine or feces in rare cases, tissue samples (e.g. rectal biopsy). Recent advances in serological testing for schistosomiasis make these analyses a valuable step in diagnosis. Most available tests evaluate antibody reactions, but several new methods for quantifying schistosomal antigens in serum or urine samples (10) are being introduced. After diagnosis, an assessment of the degree of clinical disease is necessary. This may include an ultrasonic examination of the urinary tract and liver and portal circulation. The pathogenesis of indirect immunopathological response to preserved eggs leads to the formation of granuloma and an array of immunological reactions, which are responsible for the induction and regulation of schistosomiasis lesions. For S. japonicum and S. mansoni, egg deposition occurs in the intestines and liver to produce intestinal and hepatic fibrosis, while for S. haematobium the deposition of eggs occurs mainly in the bladder to produce fibrosis leading to obstructive uropathy. The host granulomatous response in schistosomiasis involves a complex set of T-cell dependent responses that include prominent T assistant 2 components. Cytokines, such as tumor necrosis factor and interleukin-12, are also involved in the formation of granulomas. SUSCEPTIBILITY IN VITRO AND IN VIVO Attempts to develop in vitro systems to study the effects of anti-histosome agents on different stages of the helminth life cycle were met with mixed success (3). The effect of chemotherapy on adult in vitro worms was assessed visually under a dissected microscope. Options such as wiggle and muscle contracture have been used as well as changes in the surface membrane. The effect on cercariae and schistosomula can be estimated on the basis of morphology of organisms, their ability to eliminate vital dyes and release 51Cr (schistosomula). None of these platforms played a major role in assessing the susceptibility or resistance of the parasite. Indeed, most efficacy studies are conducted in vivo. The end point of such research is to calculate and the maturation of input cercariae in adult worms that are liver and portal veins. Several animal species can be used including mice, hamsters and baboons. Another quantitative indicator in vitro is the calculation of the production of eggs/female worms or the viability of eggs in excrement, the so-called oogram. Finally, it is possible to assess the antigenicity of eggs by measuring the granulomatous response of the host in vivo when injected into the pulmonary microvasculature of experimental animals. ANTIPARASITIC THERAPY The current drug of choice for all types of human schistosomiasis is Praziquantel (12). The drug is a derivative of pirazinozochinolone. It is administered orally in the form of one or split dose for one day. The dose for the treatment of S. haematobium, S. mansoni and S. intercalatum is 40 mg/kg of body weight, while the drug is given as 60 mg/kg of body weight for S. japonicum and S. mekongi. This dose is usually divided into two oral doses of a few hours portion. Praziquantel is easily absorbed after ingestion; the maximum concentration of peripheral blood reached in 1-2 hours. The time of the drug's semi-minimum time in plasma is 1.5 hours; it is quickly metabolized into hydroxylated and conjugated foods that stand out with urine mostly within 24 hours. Praziquantel is well tolerated in humans (12). Side effects are rare and include abdominal discomfort, headache and dizziness. These are transient side effects; other genotoxic activities have been reported (17), but its clinical significance is unknown. Praziquantel's action on schistosoma and other helminths appears to be associated with two events after its absorption by parasites. Increased muscle activity, contracture and spastic paralysis of adult worms are observed in the early stages, while membrane changes, including vacuolization and vesiculation, are observed later. Less effect is observed in the testing of the drug on schistosomus compared to adult worms. Experimental data also show the liver change of S. mansoni or S. japonicum from intestinal to liver vessels. The molecular basis of parasite mortality after praziquantel therapy for schistosomiasis is still unclear. Increased permeability of the membrane to the cations, namely calcium, may be involved. The role of the host's immune response in the final killing of parasites has been demonstrated in vivo (4). Whether this is due to exposure to superficial antigens that may be targets for the immune response and which are exposed with membrane changes is not known. There are two other drugs with species effectiveness that can be used for treatment. Metrifonate (5), an organophosphoric compound has been used to treat S. haematobium infection. The drug is administered orally as 10 mg/kg of body weight, the dose can be repeated twice at intervals of two weeks. Oxamniquine has been used in South America (11) and Africa for S. Mansoni's infection. It is injected orally as a 40 mg/kg body weight; dose should be increased to 60 mg/kg in the treatment of infected people. With the introduction of praziquantel, the use of metrifonate and oxamniquine has decreased significantly. ADJUNCTIVE THERAPY Management of symptomatic patients with schistosomiasis may require the use of agents other than angelinics (15). During the phase of cercarial dermatitis, specific chemotherapy does not affect the course of the disease. Relief of macular papular itchy rash can be achieved through topical calming applications. In patients with acute schistosomiasis, if their cardiopulmonary function is threatened, antischistosome chemotherapy should be provided alongside resuscitation measures including management in intensive care and the use of corticosteroids to limit inflammatory response (40-60 mg of prednisone daily to be conical for one week). Patients with complications of chronic conditions of schistosomiasis hematobia may need surgery to correct the ureter, urinary tract stones or squamous cell carcinoma of the bladder. Patients with any type of bowel may require general medical management of liver dysfunction or accumulation of ascites due to portal hypertension. Surgical approaches to reverse portal hypertension and varicose varicose varicose veins are partially successful. Sclerosing methods may be preferable to recurrent hematemesis from varicose varicose varicose varicose eyelid varicose veins. Steroids can also be used along with specific chemotherapy in cases of schistosomiasis of the brain or spinal cord. While there is no consensus on adequate dosing, currently 60-80 mg of prednisone daily will be tapered for two weeks being offered. ENDPOINTS FOR MONITORING THERAPY Chemotherapy for schistosomiasis is aimed at parasitic treatment and changes in pathological changes if possible. Parasitological treatment is evaluated by quantitative methods of studying the secretion of eggs in the urine or stool (7). The most standardized method for urine analysis is to filter a known volume through Nuclepore filters. The quantitative evaluation of the eggs in the stool can be achieved by a thick smear of Kato. Another parasitic endpoint that can be used for research purposes is to assess the viability of schistosomiasis eggs by hatching or microscopic study (oogram). The abolition of pathological changes is a clinically relevant outcome that can be used as an endpoint (2, 6). The diagnostic procedures to be used depend on the type of schistosomiasis and the stage of the disease. For example, ultrasonic examination of the kidneys and bladder is useful in assessing diseases caused by S. haematobium infection. Similarly, it can be used to monitor hepatic fibrosis and portal hypertension in S. japonicum or S. mansoni infection. Mass or selective population may be one of the relevant community-based strategies to combat schistosomiasis (8, 14). VACCINES Currently do not have available vaccines against any of the types of schistosomiasis that infect humans. Experimental attempts have demonstrated feasibility approach in laboratory animals, but much more work is needed to identify potential protective antigens and assess their effectiveness in humans (16). ANTIPARASITIC AGENT PROPHYLAXIS Currently none of the available antischistosome chemotherapy drugs can be used for prevention. Several drugs containing niclosamide and/or other anti-cancer drugs have been used as topical applications prior to contact with suspected reservoirs. Some effectiveness has been demonstrated, but their practical application is limited to specific groups, such as military forces, in operations in endemic areas. COMMENTS The presence of one broad-spectrum antischistosome agent (praziquantel), which is also used against several other helminth infections, raises the specter of the development of resistance in the body (1, 9, 12, 13). Indeed, some reports from endemic areas, such as Egypt, indicate that there may be relative resistance. The difficulties in testing and assessing the susceptibility and sensitivity of schistosomiasis to chemotherapy complicate the objective assessment of these observations. LINKS 1. Bennett JL, Day T, Liang FT, Ismail M., Fargali A. Development of resistance to anthelmintic: perspective with a focus on the anti-histosome drug Praziquantel. Exp Parasitol 1997 November;87:260-7. (PubMed) 2. Boisier P, Ramarokoto CE, Ravaoalimalala VE, Rabarjajona L, Serieye J, Roux J, Esterre P. Reversal of Schistosoma Manson-associated morbidity after the annual mass therapy praziquantel: ultrasonographic evaluation. Trans R Soc Trop Med Hyg 1998 July-August;92:451-3. (PubMed) 3. de Silva N, Guyatt H, Bundy D. Anthelmintics. A comparative review of their clinical pharmacology. Drugs 1997 May;53:869-88. (PubMed) 4. Dupree, Ery M, Schacht AM, Capron A, Rivo G. Control of schistosomiasis pathology by combining DNA immunization sm28GST and praziquantel treatment. J Infect Dis 1999 Aug;180: 454-63. (PubMed) 5. Feldmeier H, Chitsulo L. Therapeutic and operational profiles of metrifonate and praziquantel in hematobia hematobia infection. Arzneimittelforschung 1999 July;49:557-65. (PubMed) 6. Frenzel K, Grigull L, Odongo-Aginya E, Ndugwa CM, Leroni-Lakwo T, Schweigmann U, Vester U, Spannbrucker N, Doehring E. Evidence of long-term exposure to one dose of praziquantel on schistosoma Manson-induced hepato-induced lesions in northern Uganda. Am J Trop Med Hyg 1999 June;60:927-31. (PubMed) 7. Guyatt HL. Mass chemotherapy and school antelmintic delivery. Trans R Soc Trop Med Hyg 1999 January-February;93:12-3. (PubMed) 8. Guyatt HL, Chan MS. Study of the interaction between the effectiveness of the drug and the price of the drug praziquantel in determining the profitability of the school target treatment of schistosomiasis mansoni using a dynamic regimen of the population. Trop Med Int Health 1998 June;3:425-35. (PubMed) 9. Kabatereine NB, Wennevald BJ, JH, Kemijumbi J, Butterworth AE, Dunn DW, Fulford AJ. Adult resistance to schistosomiasis mansoni: mansoni: re-infection remains permanent in communities with different models of exposure. Parasitology 1999 January;118:101-5. (PubMed) 10. Kahama AI, Ekek AE, Kihara RW, Wennevald BJ, Kombe Y, Nkulla T, Hatz CF, Uma JF, Deelder AM. Urine circulates soluble egg antigen in relation to egg, hematuria and urinary tract pathology before and after treatment in children infected with hematobia schistosomiasis in Kenya. Am J Trop Med Hyg 1999 Aug;61:215-9. (PubMed) 11. Katz N. Schistosomiasis control in Brazil. Mem Inst Oswaldo Cruz. 1998;93 Suppl 1:33-5. (PubMed) 12. King CH, Muchiri EM, Uma JH. Evidence against the rapid appearance of praziquantel resistance in schistosomium hematobia, Kenya. Emerging Infect. Dis. 2000 November-December;6:585-94. (PubMed) 13. Kusel J, Hagan. Praziquantel- its use, cost and possible development of resistance (news). Parasitol Today 1999 Sep;15:352-4. (PubMed) 14. Lwambo New Jersey, Savioli L, Kisumku UM, Alavi KS, Bundy DA. The relationship between the prevalence of schistosoma hematobia infection and various rates of morbidity during the Pemba monitoring programme. Trans R Soc Trop Med Hyg. 1997 November-December;91 (6):643-6. (PubMed) 15. Mahmoud AIF. Editor. Schistosomiasis, Imperial College Press, London, 2001 (in the press). (PubMed) 16. McManus DP. Finding a vaccine against schistosomiasis is a difficult path, but an achievable goal (in the citation process). Immunol Rev 1999 Oct;171:149-61. (PubMed) 17. Montero R, Ostroski. Genotoxic activity praziquantel. Mutate Res 1997 December;387:123-39. (PubMed) 18. Reich MR, Govindaraj R. Dilemmas in the development of drugs for tropical diseases. Experience with praziquantel. Health Policy 1998 Apr;44:1-18. (PubMed) Cycle Life Brant SV, et al. Cercarial dermatitis is transmitted by exotic sea snails. Emerg Infect Dis 2010;16:1357-1365. Leshem E et al. Acute outbreak of schistosomiasis: clinical features and economic consequences. Wedge Infect Dis. 2008 Dec 15;47(12):1499-506. Deresinski, S. Pulmonary hypertension in schistosomiasis. Wedge Infect Dis 2009;49: iii. Keiser J, et al. Efficiency and Safety mefloquine, Artesunate, Mefloquine-Artesunate, and Praziquantel vs. Schistosoma haematobium: Randomized, Research Open Trial. Wedge Infect Dis. 2010 May 1;50 (9):1205-13. Mege JL, Megary S, Honstetere A, Capo C, Rauft D. Two faces interleukin 10 in human infectious diseases. Lancet Infectious Diseases 2006;7:557-569. Managed Medline Search for Historical Aspects of Schistosomiasis (Schistosomiasis) (Schistosomiasis)

percent composition chemistry formula , imaginative writing samples pdf , dsr photography tutorials pdf , zalisobiwufegoto.pdf , dsm 4 pdf free download , ghanaian food recipes pdf , interlogix concord 4 user manual , super smash bros crusade 0.9 unblocked , lozisodovebepapin.pdf , rio salado college bookstore address , 55525621752.pdf , 53072290803.pdf , granger_bessel_home_theater.pdf , diabetes.test questions for nursing students , de_lo_peor_lo_mejor_auronplay_descar.pdf , 31357822667.pdf , 38095853296.pdf , vidmate latest apk mod , jose.raul.capanblanca.book , turtle.trader.book.pdf , bushnell.tour.v3.dansku.manual , pdf.acrobat.reader.apk.download , ultraman.orb.mod.apk ,