Genitourinary Schistosomiasis. A Pictorial Review.

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Learning objectives

To summarize the spectrum of radiological findings of genitourinary lesions of Schistosoma Haematobium including abnormalities on plain x-ray, ultrasonography and CT.

To emphasize the importance of recognition of radiological findings which may aid to confirm the diagnosis, in cases of clinical suspicion and negative laboratory analysis.

To remain updated on the status of Schistosomiasis with increasing frequency in all around the world.

Fig.1 on page 2

Images for this section:

Fig. 1: Title
Background

Schistosomiasis is increasing in frequency due to shifting demographics. Clinical outcome of infection is variable, often silent, ranging from mild symptoms, anemia, scarring of genitourinary tract, renal failure and squamous cell bladder. Diagnosis is based on clinical suspicion followed by laboratory studies. However, egg production may be low and immunodiagnostic determination does not necessarily correlate with parasite load, so the risk of losing the diagnosis is high. Medical treatment has not been 100% effective in eliminating infection.

Imaging findings OR Procedure details

Introduction

The schistosomiasis, also know as bilariasis, was identified for the first time by Teodor Biliharz in 1852.

This disease is caused by a blood infection. Such infection is a result of the action of the Trematodos parasites, from the Schistosoma family. This affection can become highly chronic and end in high sickness rates and fatalities. Schistosomiasis is the second tropical parasitosis most common after malaria.

Epidemiology

Around the world there is more than 200 million people infected. 85% of the cases are in Africa, and there is more than 700 million people in risk of infection in 74 subtropical and tropical countries. In these countries the disease is endemic due to the people's contact with fresh water contaminated with snails, mostly during farming, domestic and recreational activities. Bad hygienic habits and community activities make children even more vulnerable.

The increase of intercontinental travelling and the significant raise of people migrating from endemic areas, had caused an important increase of detected cases of Shistosomiasis in developed countries during the last 20 years. These scenario is more prevalent in Europe, and specifically Spain, due to its geographical closeness to Africa.

There are two main kinds of Shistosomiasis: urogenital and intestinal, caused by 5 different kinds of blood parasites:
<table>
<thead>
<tr>
<th>Kinds</th>
<th>Geographic Distribution</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Urogenital Schistosomiasis</strong></td>
<td>Northern Africa</td>
</tr>
<tr>
<td><em>S. hematobium</em></td>
<td>Subsaharan Africa</td>
</tr>
<tr>
<td></td>
<td>Middle East</td>
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<td></td>
<td>Turkey and India</td>
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<tr>
<td><strong>Intestinal Schistosomiasis</strong></td>
<td>Subsaharan Africa</td>
</tr>
<tr>
<td><em>S. mansoni</em></td>
<td>Middle East</td>
</tr>
<tr>
<td></td>
<td>The Caribbean, Brazil and Indonesia</td>
</tr>
<tr>
<td><em>S. japonicun</em></td>
<td>China</td>
</tr>
<tr>
<td></td>
<td>Philippines</td>
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<tr>
<td><em>S. mecongi</em></td>
<td>Indonesia y Thailand</td>
</tr>
<tr>
<td><em>S. intercalatum, conjenere S. guineasis</em></td>
<td>Laos</td>
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<td></td>
<td>Camboya</td>
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<td></td>
<td>Vietnam</td>
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<tr>
<td></td>
<td>Only for West and Central Africa</td>
</tr>
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The World Health Organization (WHO) reports that around 70 million people suffer urinary Schistosomiasis, 18 million from illness associated with the wall of the urinary bladder, and 10 million of hydronephrosis. The estimated deaths caused by kidney failure related are around 150,000 people, and an unknown important number are due to urinary bladder cancer and other genitourinary neoplasms. Fig. 1 on page 11

### Life Cycle

To know the life cycle of the parasite is crucial because it let us understand the different stages of the disease, and the evolution of the clinic. An encompassing knowledge of the parasite and its actions give us a better picture to interpret and read the different studies, hence a more certain diagnose.

Humans are the ultimate hosts for the parasite's eggs. The eggs are introduced and spread to aquatic systems through human's feces and urine, mostly when people does
their biological necessities close to rivers or wells. After some incubation time, eggs with embryos inside hatch into miracidia.

The miracidia can swim in fresh water up to 32 hours, then it penetrates the snail, which is its intermediary host. Inside the snail the miracidia breeds asexually into sporocysts, which later become cercariae larvae; being these last the ones that infect the human being.

Cercarie could survive up to 48 hours before they find their ultimate host. They attach themselves to healthy skin and can trespass it using its proteolitic enzymes. At this stage of the invasion they can cause a burning sensation and temporary skin wounds, and it's at this point that they lose their tail and become into shistosomula. Once they trespass the skin, shistosomula travels to the blood.

Vessels and to the lymphatic system. From there they reach the porta system, right heart and lungs, where they continue their life cycle. Then they return to the capillaries, left heart, and from the arterial circulation they reach the liver. Once in the liver shistosomula establish at the hepatic sinusoides where they feed and breed til they reach maturity after 4 weeks. During this incubation period the infected person can suffer of minor hemorrhage, fever, eosinophilia and leucositosis.

The different species of shistosomula differ in their preference for a special kind of organ tissue. Adult worms migrate to the vesical venous plexuses (S. hematobium), to mesenteric venules of the small intestine (S. japonicum y S. mekongi), colon's mesenteric venules (S. mansoni y S. intercalatum), living in the blood vessels, easily surviving from 5 to 7 years, and in extreme cases staying for as much as 30 years.

There, females live in the gynecophoric canal of the human male and lay down eggs, from 300 to 3000 a day. At this stage of the infection hematuria stars, and also some bloody feces. Eggs travel through hematogena to other places or they can pass from the vascular area to the light of the intestine or the urinary bladder where they can produce an inflammatory reaction. Repair of the tissues also stars, and granulomas and fibrosis begin to show up. Eggs are expelled through urine and feces and as soon as they get in contact with fresh water they star a new life cycle. Fig.2 on page 12

**Pathophysiology and clinical manifestations**

The pathophysiology of schistosomiasis involves the immune response against the schistosome eggs. The clinical manifestations depend on the species of parasite, intensity of worm burden, and immunity of the person to the parasite.
Humans are estimated to excrete approximately 50% of the eggs. The rest are trapped in various parts of the body. Occasionally, the worm can be in ectopic positions, such as in the spinal cord, where it produces unusual clinical manifestations.

The pathophysiology correlates with the life cycle of the parasite:

- Cercarie: Skin penetration of cercariae produces an allergic dermatitis at the site of entry. With prior sensitization, a pruritic papular rash develops. This also is observed with nonhuman avian schistosomes.
- Schistosomula: These are tailless cercariae that are transported through blood or lymphatics to the right side of the heart and lungs. Heavy infection can cause symptoms such as cough and fever. **Eosinophilia may be observed.**
- Adult worm: Adult worms do not multiply inside the human body. In the venous blood, adult male and female worms mate, and the female lays eggs 4-6 weeks after cercarial penetration. **Adult worms are rarely pathogenic.** The female adult worm lives for approximately 3-8 years and lays eggs throughout her life span.
- Eggs: **They cause Katayama fever and schistosomiasis**

- Katayama fever: The exact pathophysiology is not known. It occurs **4-6 weeks after infection**, at the time of the initial egg release. It is reported most commonly with *S. japonicum*. It’s believed to be due to the high worm and egg antigen stimuli that result from immune complex formation and lead to a serum sickness-like illness. Fever, lethargy, and myalgia are the most common symptoms.

- **Schistosomiasis:** It is due to immunological reactions to *Schistosoma* eggs trapped in tissues. Antigens released from the egg stimulate a granulomatous reaction comprised of T cells, macrophages, and eosinophils that results in clinical disease. Symptoms and signs depend on the number and location of eggs trapped in the tissues. Initially, the inflammatory reaction is readily reversible. In the latter stages of the disease, the **pathology is associated with collagen deposition and fibrosis, resulting in organ damage that may be only partially reversible.**

- **Urinary tract schistosomiasis:** Asymptomatic or may cause microscopic or macroscopic hematuria, terminal hematuria, dysuria, and urinary frequency. Symptoms related to secondary anemia may be present. This can lead to renal failure due to obstructive uropathy, pyelonephritis, or bladder carcinoma (occurring usually 10-20 y after the initial infection). In addition, immune complexes that contain worm antigens may deposit in the glomeruli, leading to glomerulonephritis and amyloidosis.

- **Female genital schistosomiasis (FGS):** *S. haematobium* causes lesions in the female lower genital tract (ie, cervix, valva, vagina). FGS has been identified as a major social and medical problem that may facilitate the spread of some sexually transmitted diseases
such as HIV and human papillomavirus (HPV) and infertility. Type of genital lesions: perisalpingitis and interstitial salpingitis as result of ova deposition. The ovary may be involved and bilharzial ova found in hilum Schistosomiasis of the vulva. Polypodial mass in vagina.

- **Male genital schistosomiasis**: haemospermia and lumpy semen. Calcifications of the seminal vesicles and prostate. There are a small number of case reports of lesions of schistosomiasis developing within the testes (simulating carcinoma or causing infarction), epididymis, and the penis. Male infertility resulting from such lesions is rare. fig 12 on page 22. fig 13. on page 23 fig 14. on page 24

**Laboratory Studies**

- Stool or urine analysis
  - Identify and speciate the eggs in the stool or urine.

Urinary excretion of eggs is not uniform. The urine is most likely to be positive for *S hematobium* from 10 am until 2 pm

- Acute illness is often associated with eosinophilia in the blood and tissues. With chronic illness, peripheral eosinophilia may be minimal or absent while tissue eosinophilia persists.
- Urinary schistosomiasis (occurs with chronic disease)
  - Urine syringe filtration techniques provide a quantitative estimate of eggs in the urine.
  - Urine analysis and culture for hematuria, proteinuria, leukocyturia, and associated urinary infections
- Serology
  - Detecting antibodies specific to *S mansoni*, *S hematobium*, and *S japonicum* adult worm microsomal antigens.
  - The finding of calcified eggs in the seminal vesicles, or in the female genital tract is also a strong indication of schistosomiasis.

**Procedures**

- Rectal biopsy or bladder mucosal biopsy
  - Mucosal biopsy is effective for visualizing eggs.
  - Biopsy is helpful when stool sample findings are negative or in light infection.
  - Obtain multiple biopsy samples and crush them between slides (to increase egg-detecting sensitivity).
- Cystoscopy
  - To obtain mucosal biopsy for diagnosis
• To assess complications such as bladder cancer

**Imaging findings**

- Imaging findings mirror the pathologic course. With acute schistosomiasis, a chest radiograph sometimes demonstrates a generalized increase in vascular and interstitial marking and mild lymphadenopathy.
- In the **acute phase**, nodular bladder wall thickening is observed at urography or cross-sectional imaging. The earliest change in the ureters is persistent filling of the lower segment in all films of the urography series. At this stage the ureters are not dilated or otherwise abnormal in many patients, but dilatation of the filled distal, intrapelvic segment is the next change. The widening may be slight or marked, but there may be no visible stenosis. The earliest ureteric constriction is within the bladder wall, and there will also be changes in ureteric peristalsis. This has been confirmed by fluoroscopic studies.
- **The chronic phase is characterized by a contracted, fibrotic, thick-walled bladder with calcifications.** These calcifications are typically curvilinear and represent the large numbers of calcified eggs within the **bladder wall**. Distal ureteral calcification may also be present. A mass may be secondary to inflammation or complicating carcinoma, typically squamous carcinoma. Other ureteric complications of schistosomiasis include pseudo tubercles in the ureters, ureteric stenosis and strictures resulting on proximal ureteric dilatation and varying degree of hydronephrosis as well as ureteric calculi. A late sequale of ureteric involvement is ureteric calcification commonly involving the lower third ureter and middle third and less so the upper third of the ureter.

- **Plain abdominal radiographs may demonstrate bladder and ureteral calcifications.**

Plain radiography of the abdomen is of no assistance until calcification has developed in the bladder and ureters, although in severe infections calcified eggs may also be seen in the soft tissues. (Early calcification is much more clearly seen by CT.) Intravenous (contrast) urography will provide very useful diagnostic information. **All the early findings will be in the ureters and bladder; the kidneys remain normal until later in the disease.**

The important radiological findings in the urinary tract are as follows:

- **A. In plain films and computed tomography fig. 3 on page 13**
1. Parallel lines of calcification following the contours of the collapsed empty bladder. fig. 4 on page 14

2. A single thin line of calcification outlining the periphery of the distended bladder. fig. 5 on page 15

3. Less commonly, similar calcifications in the lower ureters.

   - **B. In the excretion pyelogram**

   1. Ureteral stasis, going on eventually to stasis plus dilatation of the lower spindle, and eventually of the whole ureter and pelvi-calyceal system.

   2. Bladder filling defects.


   - **On a sonogram**, hydronephrosis, hydroureters, and bladder wall irregularities may be visible. Can detect hypertrophy of the bladder mucosa, thickening of the bladder wall (which is normally less than 5 mm when the bladder is distended), and bladder calcification. If the ureters are easily demonstrated by ultrasound, they are probably thickened and abnormal; in some patients calcification will be seen, but ultrasound is not a reliable way to demonstrate early ureteric changes. Dilatation of the renal collecting system can be detected very early, and represents the result of significant ureteric dysfunction. fig 6. on page 16 Fig 9. on page 19 Fig 10. on page 20 Fig 11. on page 21

   - Because of the strong association between schistosomiasis haematobium and squamous cell carcinoma of the bladder, particular care should be taken during ultrasonography to accurately record bladder wall thickening. **Repeat ultrasonography is advisable after treatment; about 70% of schistosomal bladder lesions will regress significantly in less than 12 months (after treatment with praziquantel).** If a bladder lesion does not improve, long-term follow-up is required to exclude malignancy. Over 90% of patients who have schistosomal carcinoma of the bladder will have ureteric obstruction; in a non-schistosomal (transitional cell) bladder carcinoma less than 25% will have ureteric obstruction. fig 7. on page 17 fig 8. on page 18

**Differential Diagnosis (S. haematobium)**

The differential diagnosis of calcification of the bladder is not difficult in most patients because each cause has a very different appearance.
Calcification can be due to:

1. **Amyloidosis**, in which it is very spotty.

2. **Some chemicals and drugs**, such as cyclophosphan may cause quite heavy calcification, but the bladder will be small and does not expand.

3. **Tuberculosis** can cause localized calcification in one, or perhaps, two areas of the bladder, usually a small flat area.

4. **Carcinoma** which has been treated by radiotherapy may also calcify when it heals, associated with scarring and distortion of the bladder outline and localized to one area. There is nearly always residual local thickening.

In schistosomiasis the bladder calcification follows the shape of the bladder, is seldom associated with distortion except in the final stages, and does not usually affect bladder distention or contraction.

**It is by far the commonest cause of bladder calcification and is very characteristic. It is the only cause of bladder calcification in which there is likely to be calcification of the ureters as well.**

In those who have acquired the infection recently (such as travelers) as opposed to those who have lived with it for many years, it is important to recognize the early signs of prolonged ureteric filling during contrast urography. This may be the only evidence of the infection. Later the irregularity and beading of the ureter, the stenosis and dilatation can be recognized by ultrasound or radiography at a time when the bladder may still be normal.

**Treatment**

The aim of chemotherapy is 2-fold. The first goal is to cure the disease or at least minimize morbidity. The second goal is to control transmission of the parasite in the endemic areas. Praziquantel (Biltricide) is used commonly, which destroys the adult worms and incites the eggs to hatch. It has no effect on the chronic fibrotic changes in the bladder wall and ureters. But praziquantel is the treatment of choice for all species of schistosomiasis. Cure rate is equal to or greater than 85%. In persons not cured, the egg burden is markedly decreased.

Response to treatment is evaluated by counting the amount of decrease in egg excretion. Persistent circulating antigen and the excretion of eggs indicate residual infection. These patients should be retreated with praziquantel.
Following treatment, particularly in those who have not been previously infected, follow-up ultrasound and intravenous urography after six months will ensure that there has been no ureteric stenosis or dilatation.

**Schistosomiasis and bladder cancer**

A large and compelling body of evidence links schistosomiasis of the urinary tract to bladder cancer. The mechanisms involved are not well understood, and many different etiological factors could be involved. Schistosomiasis induces chronic irritation and inflammation in the urinary bladder, and this could facilitate changes in at least two stages of the development of the disease: first, initiation of premalignant lesions, and second, action as a promoting agent to increase the likelihood of the conversion of these lesions to the malignant state.

In recognizing this sequence of events, it is fairly clear how prevention could be achieved. Elimination of the parasite through education, improved hygiene, and improved conditions for living and working are the obvious solutions, but the level of investment required for this is well beyond the resources of most of the countries where infection is endemic. A possible interim solution that requires less extensive financial resources is chemotherapy with effective agents such as praziquantel; however, this approach is realistic only when individuals move away from areas where the parasite is prevalent and hence from sources of reinfection.

Extensive analysis of the results of 9 case-controlled studies showed that risk factors for nontransitional-cell bladder carcinoma (NTCC) included toxic compounds (eg, pesticides), smoking, and excessive alcohol consumption. Egypt is known to have the highest bladder cancer rate in the world. Investigation of the link between development of NTCC and schistosomiasis, an infection with a high prevalence rate in this country, revealed that 45% of patients with NTCC and 37% of controls had been infected. Schistosomiasis may be associated with a higher risk of developing NTCC, and according to this study, it may be responsible for about 16% of all bladder cancers in Egypt.

**Images for this section:**
Fig. 1: fig.1
Fig. 2: fig.2
Genitourinary Schistosomiasis

Imaging Findings-Plain x-ray

Patient with a history of recurrent urinary tract infections, bleeding in the urine, eosinophilia, negative parasites in urine and liver, which in the radiograph shows linear calcification in right ureter and urinary bladder (arrow).

Fig. 3: fig.3
Patient 33 years old from Mali with positive urine for schistosomiasis which in the CT shows calcification of the bladder wall.

Fig. 4: fig.4
Genitourinary Schistosomiasis

Imaging Findings-CT

Patient 24 years old from Guinea. CT shows linear calcification on the bladder urinary wall.

Fig. 5: fig.5
Genitourinary Schistosomiasis

**Imaging Findings-Ultrasound-CT**

Patient 17 years old form Gambia, with positive urine for schistosomiasis. Ultrasound shows nodular thickening in the right lateral wall of the urinary bladder (arrows), after treatment nodular swellings disappears. CT shows linear calcification of urinary wall bladder.

**Fig. 6:** fig 6
Patient 59 years old with history of urinary schistosomiasis. In the ultrasound show a solid mass on urinary bladder, well defined and lobed of contours, displacing the bladder catheter.

Fig. 7: fig 7.
Fig. 8: Patient 59 years old with history of urinary schistosomiasis. Axial CT shows a solid mass on urinary bladder, well defined and lobed of contours, displacing the bladder catheter.
Fig. 9: Fig 9.

Patient 34 years old, with eosinophilia that in the ultrasound show left ureterocele. (arrow)
Patient 20 years old with history of urinary schistosomiasis, eosinophilia that in the ultrasound shows hyperechoic focal thickening (arrow), on the side walls and bladder floor.

Fig. 10: Fig 10.
Patient 15 years old with history of urinary schistosomiasis. Ultrasound shows in the anterior region of the dome of the left urinary bladder image iso-hyperechoic with a base of 13 mm and a thickness of 7 mm. (arrow)

**Fig. 11:** Fig 11.
Scrotal schistosomiasis in a 26-year-old man with long-term scrotal swelling. (a) Axial US and CT image shows a septate fluid collection and hydrocele. Punctate calcifications are seen within a thickened tunica vaginalis (arrows) and curvilinear calcification of bladder wall.

**Fig. 12:** fig 12
Genitourinary Schistosomiasis

Imaging Findings-Ultrasound

Scrotal schistosomiasis in a 28-year-old man with long-term scrotal swelling. Axial US image shows a septate fluid collection. Punctate calcifications are seen within a thickened tunica vaginalis (arrows).

Fig. 13: fig 13
Scrotal schistosomiasis in a 28-year-old man with long-term scrotal swelling. (a) Axial US image shows a septate fluid collection and hydrocele. Punctate calcifications are seen within a thickened tunica vaginalis (arrows). (b) Photomicrograph shows worm eggs (arrows), an inflammatory fibrotic reaction, and calcifications.

**Fig. 14:** fig 14
Conclusion

This work provides a comprehensive overview of the spectrum of genitourinary imaging findings by Schistosoma Haematobium.

Knowledge of radiologic presentation of schistosomiasis may contribute to the diagnosis and give an indication of disease severity by demonstrating complications.

Personal Information

References


