Clinical Imaging 54 (2019) 163–171

Contents lists available at ScienceDirect

Clinical Imaging

journal homepage: www.elsevier.com/locate/clinimag

Body Imaging

Multisystem imaging review of human schistosomiasis: characteristic imaging findings

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ARTICLE INFO

Keywords:
Schistosomiasis
Multisystem
Imaging review

ABSTRACT

Human schistosomiasis is one of the major tropical/subtropical parasitic diseases with significant morbidity and mortality. Although, the majority of the cases are seen within the endemic region, upsurge in recent immigration as well as frequent travel to endemic areas allows cases reported worldwide. Thus, it is important for radiologists and physicians be familiarized with the imaging features of schistosomiasis. Human schistosomiasis affects multiple target organs among which hepatosplenic, gastrointestinal and genitourinary organs are common. Rarely does it also affect pulmonary, central nervous system, testes etc. This article presents a comprehensive review of the characteristic imaging findings of schistosomiasis involving multiple target organs. The typical imaging findings are thoroughly correlated with the pathophysiology of parasite in human body. In addition, we have emphasized the key learning points to differentiate it from close differentials.

1. Introduction

Schistosomiasis, also known as bilharziasis is a multisystem infectious disease caused by blood fluke (trematode) of genus Schistosoma. The name 'bilharzia' is derived from the German physician Theodor Bilharz, who first described the disease after he performed autopsy in 1851 in Egypt [1]. It is the second most common tropical/sub-tropical disease after malaria. In 2015 it was estimated that Schistosomiasis affects about 250 million people around the world [2–4]. Majority of incidence (85%) is in Sub-Saharan Africa, where it causes 200,000 deaths annually [5,6]. Considerable morbidity also results from gastrointestinal, genitourinary and hepatic complications as well as from malignancy arising from urinary bladder, liver and rectum. Despite these figures, WHO mentions that the global burden of this disease is underestimated. Thus it is included under neglected tropical disease. It also affects Middle East, South America, Caribbean and Southeast Asia. Center for Disease Control and Prevention (CDC) mentions schistosomiasis is not endemic in the US [3]. However in the context of immigration from the endemic regions and US residents traveling to endemic regions, there are emerging evidences that cases of schistosomiasis even exist in the US. At least 400,000 individuals are estimated to be infected in the US, which includes immigrants, military and civilian contractors [7].

2. Discussion

Several species of blood flukes are present; *S. haematobium*, *S. japonicum*, *S. mansoni*, *S. guineensis*, *S. intercalatum*, and *S. mekongi* [8]. Among the six species, earlier three species are more common. *S. haematobium* infection is predominant in Middle East and Africa, *S. mansoni* is found in Middle East, Africa and South America. Only *S. mansoni* species is found in South America. *S. japonicum* is present in China, Indonesia and the Philippines [9]. The difference in geographic distribution of different species depends on the biodistribution of intermediate host snail. *S. mansoni* and *S. japonicum* reside in mesenteric venules and affect the liver and portal venous system, whereas *S. haematobium* resides in perivesicular venules and affects the urogenital system [10].

Life cycle.

The cycle starts as the female Schistosoma, colonizing in the

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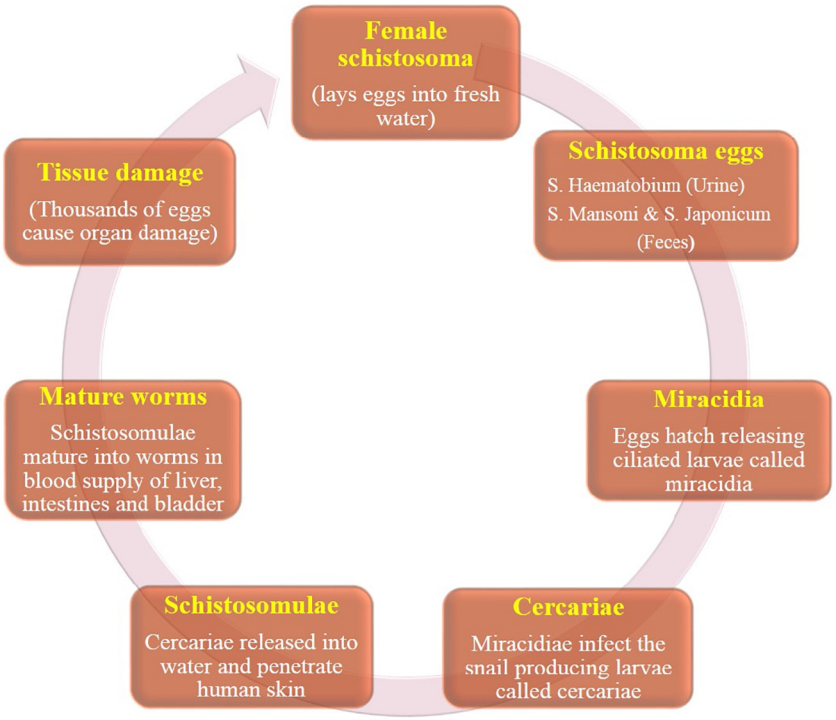
https://doi.org/10.1016/j.clinimag.2019.01.011
Received 29 October 2018; Received in revised form 7 January 2019; Accepted 15 January 2019

Table 1
Life cycle of human schistosomiasis.



infected host veins, lays the eggs that are excreted in the human urine (for *S. haematobium*) and feces (*S. mansoni* and *S. japonicum*). The eggs hatch into ciliated miracidia, which infect the tropical freshwater snails (intermediate host). Inside the snails, miracidia reproduce asexually to infectious larval form, the cercariae. They are excreted from snails, and now the free swimming cercariae can penetrate human unbroken skin. Then they can migrate via lymph or blood to different body parts, and clinical manifestations depend on the specific organ involvement. They colonize in the veins and reproduce sexually to produce multiple eggs that are either trapped in the target organs or excreted. And the cycle goes on (Table 1).

2.1. Clinical features

The clinical manifestations depend on three overlapping phases: 1. Acute stage (cercarial dermatitis, acute schistosomiasis) 2. Established active infection, and 3. Late chronic infection [11]. At skin entry site, some larvae die and incite local cutaneous hypersensitivity reaction leading to maculopapular pruritic rash known as cercarial dermatitis. Next, systemic hypersensitivity develops in response to the antigen released during parasite migration and egg release, which is characterized by fever, myalgia, fatigue, abdominal pain, diarrhea, eosinophilia, pneumonia, etc. This acute symptomatic phase is also called Katayama fever/syndrome. Both the acute phases are seen in travelers who get infected in the endemic regions. The latter two phases (established active and late chronic) are mainly seen in people living in endemic regions. The eggs deposited in the target organs provoke granulomatous reaction and fibrotic changes causing significant pathological changes in the affected organs. The severity of clinical symptoms depends on worm load and species. *S. japonicum* is more virulent than *S. mansoni* because S japonicum lays ten times the eggs as *S. mansoni* do [12]. Most commonly they affect liver, portal system, intestines and the urogenital system. Rarely brain, spinal cord, breast, genitals, eyes and skin are affected.

Diagnosis is based on epidemiologic data, clinical presentation, positive serology or demonstration of parasite eggs in stool and urine, or rectal biopsy however with their own specific limitations. Imaging with ultrasound, CT scan and MRI is not only to aid in diagnosis but also to evaluate the disease severity and complications. The radiologists are required to be familiar with the common and uncommon imaging features of schistosomiasis for accurate diagnosis and timely intervention.

2.2. Hepatosplenic Schistosomiasis

The worms in the mesenteric venules release hundreds to thousands egg per day. These eggs will be carried by blood through portal system into the liver, where they lodge at presinusoidal portal vein branches. *S. mansoni* eggs are larger than *S. japonicum* eggs, thus *S. mansoni* eggs gets lodged in larger portal vein branches near the hepatic hilum versus *S. japonicum* eggs lodged in peripheral subcapsular portal vein branches. This is the basis of central periportal fibrosis in *S. mansoni* versus peripheral periportal fibrosis in *S. japonicum* [9]. The eggs incite granulomatous reaction, leading to hepatomegaly, which progresses to chronic periportal fibrosis (Symmers fibrosis) and thus resultant portal hypertension [13]. However *S. mansoni* eggs do not calcify, but *S. japonicum* eggs do calcify. Thus calcification is predominantly seen in *S. japonicum*, but not in *S. mansoni* [12]. Interestingly, the inflammation does not involve the hepatic lobules; thus lobular architecture is preserved (with no hepatocyte destruction) and liver enzymes are normal [11,14]. The fibrotic areas subsequently undergo dystrophic calcification. *Schistosoma mansoni* infection hastens the dysplastic changes within the liver parenchyma in the presence of other risk factors, most commonly HCV infection [15].

2.3. Imaging modalities

2.3.1. Ultrasound

USG is frequently used as the first imaging modality in hepatosplenic pathologies. USG can show periportal fibrosis, left liver lobe hypertrophy and atrophy of right lobe, mosaic liver pattern, with echogenic septa outlining polygonal areas of relatively normal liver

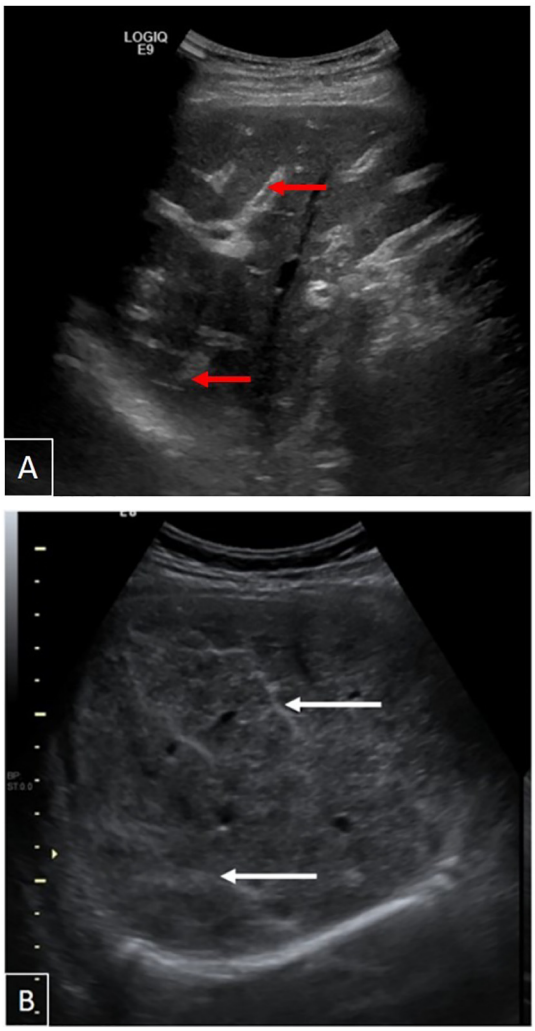


Fig. 1. Ultrasound findings of hepatic Schistosomiasis. A) Greyscale sagittal views of liver parenchyma demonstrate increased periportal echogenicity "pipestem fibrosis" (red arrows). B) Lacy pattern of liver parenchyma with polygonal echogenic septa (white arrows) outlining liver parenchyma. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

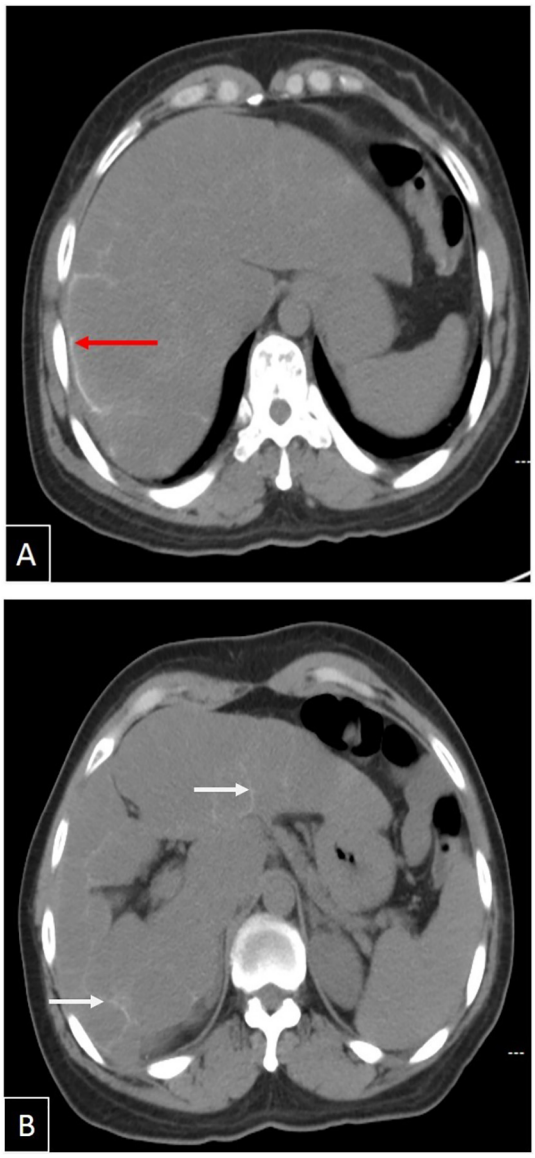


Fig. 2. Characteristic CT findings of hepatic Schistosomiasis. A) Non-contrast CT images of the liver show capsular calcifications (red arrows) (B) septal calcifications (white arrows) outlining the polygonal liver parenchyma characteristically described as "turtle back calcifications". (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

parenchyma and gallbladder wall thickening (Fig. 1). Lacy pattern of liver parenchyma with polygonal echogenic septa outlining normal liver parenchyma is specific feature of schistosomiasis. Granulomas per se are not seen directly by USG due to the small size. However rarely, they can be big enough to be seen as focal liver nodules [14]. Features of portal hypertension are seen as dilated portal venous system, splenomegaly with multiple splenic siderotic (Gamma-Gandy bodies) nodules, dilated collateral porto-systemic channels and portal vein thrombosis. Periportal fibrosis is seen as echogenic cuffing around the portal vein branches [16]. It appears as hyperechoic tubular "pipestem" longitudinally and echogenic "bull's-eye" transversely. The "bull's-eye" pattern is due to central hypoechoic portal vein and surrounding hyperechoic periportal fibrosis. Ultrasound has been used to grade periportal fibrosis as: Grade I = 3–5 mm, Grade II ≥ 5–7 mm and Grade III ≥ 7 mm [17]. This grading correlates with the disease severity in terms of portal vein dilatation, splenic vein dilatation and endoscopic variceal grade [17]. This finding can be differentiated from periportal edema in acute hepatitis. In schistosomiasis, periportal echogenicity is chronic and thicker (sometime reaching upto 2 cm) and with different clinical presentation, most remarkably jaundice being absent [14]. Other ultrasound findings such as echogenic wall thickening of the gall bladder with external wall protuberances have been described. Contrary to other parasitic diseases that infest liver, schistosomiasis does not usually obstruct biliary tree [18]. Transient elastography may be used to measure liver stiffness for assessment of liver fibrosis in patients with advanced schistosomiasis [19].

2.3.2. CT

A peculiar pattern of calcification has been described in *S. japonica*. Capsular calcification and parenchymal calcification perpendicular to liver capsule resembling septal calcification gives rise to characteristic "turtle back" or "tortoise-shell" appearance (Fig. 2). The pattern of calcification is again similar to the lacy or mosaic liver pattern seen on ultrasound which is due to the calcified parasite in periportal region The inflammation does not involve the hepatic lobules; thus preserving the lobular architecture. A dip in liver contour at the junction of capsular and septal calcification creates pseudo-lobar appearance [20]. Periportal fat can extend deep into the liver due to fibrosis with

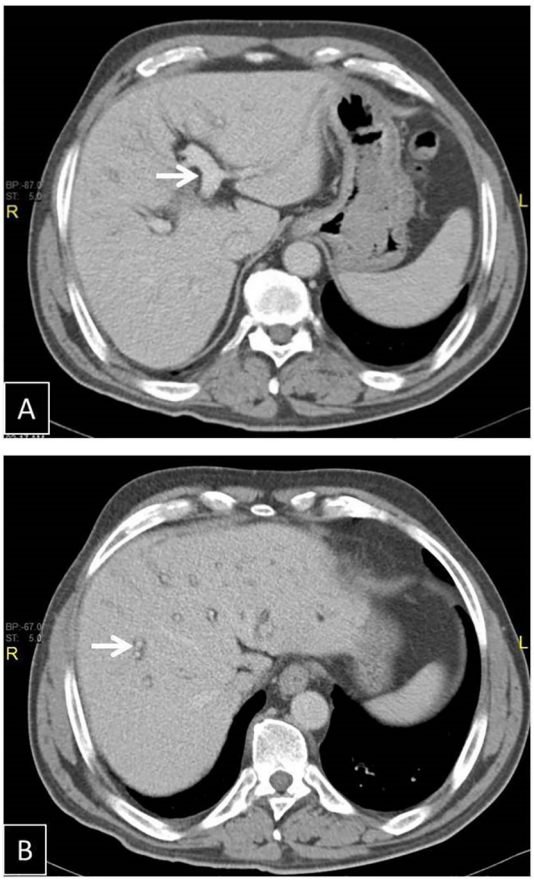


Fig. 3. Periportal fibrosis on CT scan. A,B) Contrast enhanced CT images of the liver show diffuse periportal low attenuating areas outlining both sides of portal venous radicles(white arrows).

parenchymal retraction. On CT, periportal fibrosis is seen as low-attenuation rings or thick band of hypodense tissue around the portal vein branches throughout the liver- centrally in *S. mansoni* and peripherally in *S. japonicum*. The periportal fibrosis strongly enhances in delayed venous images (Fig. 3). Features of portal hypertension can also be easily evident on CT scan.

2.3.3. MRI

MRI is sensitive modality that shows almost all the possible findings in hepatosplenic schistosomiasis except calcifications [21]. Periportal fibrosis is T1 hypointense, T2 hyperintense, and enhances strongly on post-contrast delayed venous phase (Fig. 4). T2 signal is higher in active inflammatory phase reflecting periportal edema versus relatively less higher in chronic periportal fibrosis. Thus MRI provides useful information about the acute versus chronic stage of disease, which is not possible with other imaging modalities [22]. The liver shows irregular contour with heterogeneous signal intensity and enhancement. Other features of portal hypertension can be evident as well. Splenic siderotic nodules appear hypointense in all phases including post-contrast sequences (Fig. 5).

2.4. Cirrhosis vs Schistosomiasis

Discrimination between cirrhosis and chronic hepatosplenic schistosomiasis is important because the treatment and prognosis differ. Chronic schistosomiasis can present as pseudo cirrhosis pattern on imaging [23]. Findings present in both cirrhosis and chronic schistosomiasis are heterogeneous liver parenchyma, periportal thickening, widening of hepatic fissures, and periportal venous collaterals

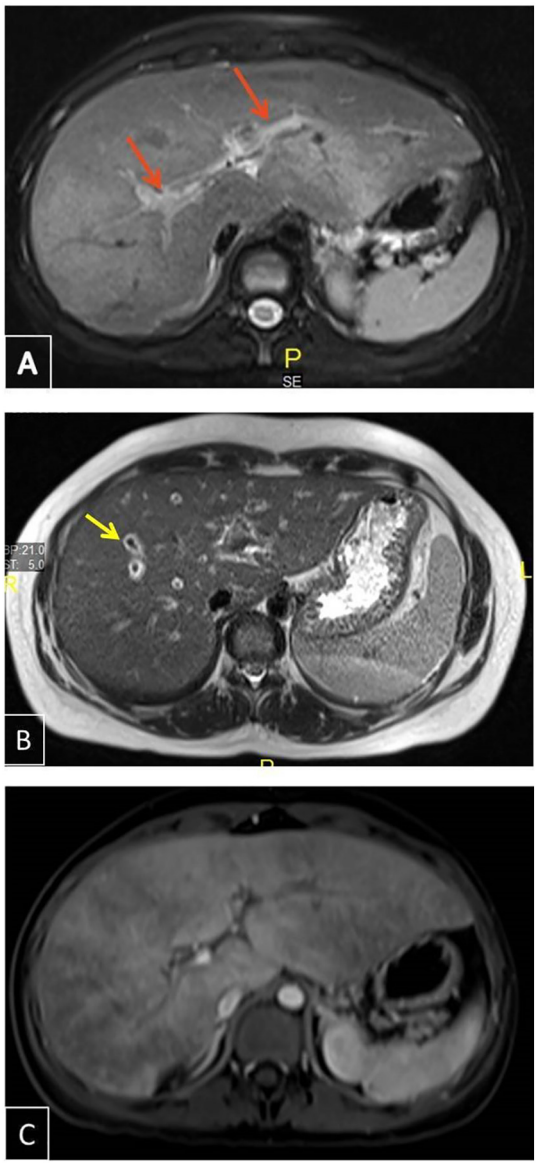


Fig. 4. Periportal fibrosis on MRI abdomen. A) Axial T2 fat saturated image shows hyperintense band like signal around the porta (red arrows) B) Non-fat saturated axial T2 weighted image shows clear visualization of periportal edema (yellow arrow).Periportal edema causing cuffing an early but non-specific feature of hepatic Schistosomiasis. C) Axial T1 weighted post contrast image of liver demonstrates hepatomegaly and heterogeneous parenchymal enhancement. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

(Table 2). Strikingly surface nodularity and regenerative nodules are absent in schistosomiasis. Periportal fibrosis, caudate lobe hypertrophy, splenomegaly and splenic siderotic nodules are more severe in schistosomiasis [24].

2.5. Intestinal Schistosomiasis

Although both the small and large intestines may be involved, adult worms most commonly inhabit the branches of the inferior mesenteric vein and superior hemorrhoidal vein. Hence, most often the eggs are deposited in the rectum, sigmoid, and descending colon [25,26]. In the inferior mesenteric venous plexus, worms lay eggs that are mainly deposited in distal colonic and rectal submucosa/subserosa. The eggs cause granulomatous inflammation, which initially result into inflammatory pseudopolyps, ulceration, bleeding and later into fibrosis,

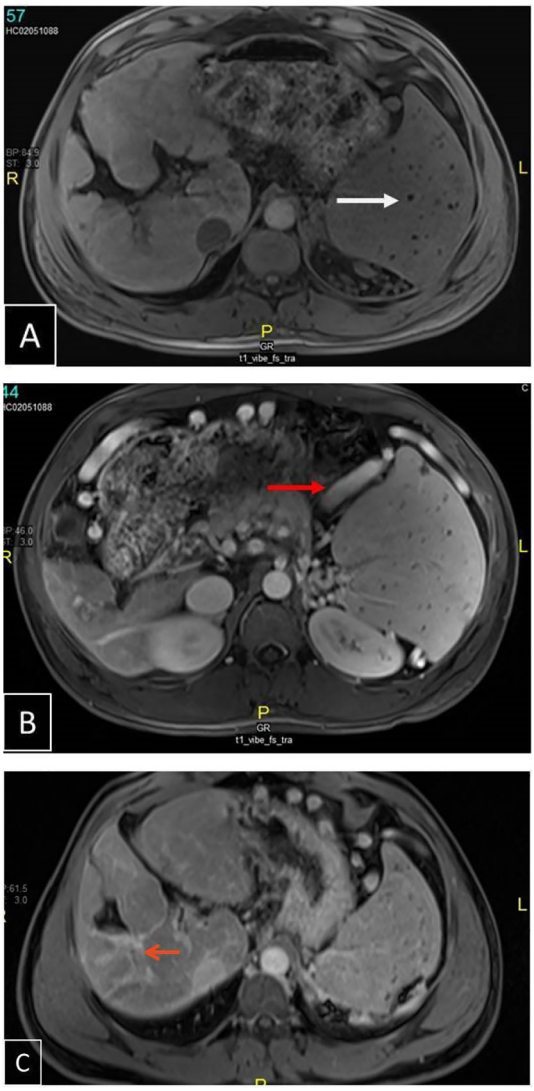


Fig. 5. MRI findings in chronic hepato-splenic Schistosomiasis. a) Axial T1 weighted non-contrast image shows shrunken liver parenchyma with atrophy of right lobe, irregular outline, widening of fissures and splenomegaly. Multiple tiny siderotic nodules are seen within the splenic parenchyma (white arrow) which are dark on all sequences b) Contrast enhanced T1 weighted axial image demonstrates porto-splenic venovenous collaterals (red arrow) identical to liver cirrhosis c) Axial post contrast image shows enhancement of the septa, capsular and periportal region (red arrow). (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

Table 2
Difference between Chronic schistosomiasis and liver cirrhosis

	Chronic Schistosomiasis	Liver cirrhosis – non specific
Periportal fibrosis	Peripheral	Central
Caudate/Right lobe ratio	Higher > 0.65	High but less than schistosomiasis
Splenic index	Higher	High
Splenic siderotic nodules	Consistent	–
Surface nodularity/ Regenerative nodules	Rare	Common feature

mural thickening, stenosis and calcification [27]. The eggs pass with stool frequently in early phase, but very infrequently in chronic phase. Thus diagnosis with stool ova visualization is easier in early phase, but difficult in later phase. Instead later phase may warrant rectal or colonic biopsy to see for the ova [28]. It may be asymptomatic or symptomatic with abdominal pain, anorexia, diarrhea or dysentery. On later stages, it may present with colonic stenosis or intestinal obstruction [27]. The polyposis does not appear to predispose to colon cancer. Mesorectal fat hypertrophy can be seen with chronic proctitis.

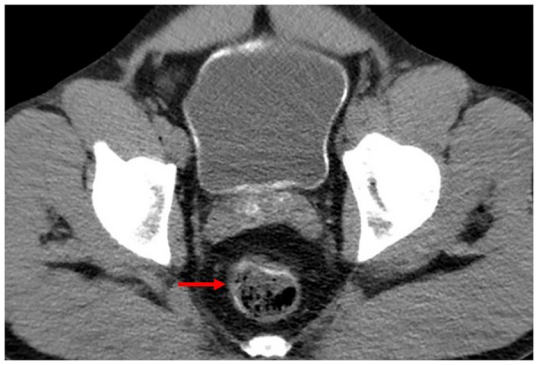


Fig. 6. CT findings in intestinal Schistosomiasis. Axial non-contrast CT image demonstrates rectal calcifications (red arrow). (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

CT demonstrates descending and sigmoid colonic calcification in curvilinear, tram-track or nodular distribution. Tram-track pattern is due to egg deposition and subsequent calcification in the submucosa and subserosal layers [29] (Fig. 6). Extensive curvilinear and tram-track calcification is characteristic of schistosomiasis. To optimally visualize the calcification, water enema is used instead of oral contrast. Water enema adequately distends the colonic lumen and reduces the air-bowel wall interface artifact. Unlike oral contrast, water enema does not mask or mimic the colonic wall calcification [30]. Other causes of colonic mural calcifications include phlebosclerotic colitis, hyperphosphatemia, renal failure and mucinous adenocarcinoma [31]. Cases of schistosomal appendicitis have been reported in literature [29,32]. CT scans are now frequently done in patients to evaluate acute abdomen, careful evaluation for appendicular mural calcification may provide a diagnostic clue.

2.6. Genitourinary Schistosomiasis

S. haematobium is the only Schistosoma species that affects the genitourinary system [32]. The parasites reside in the pelvic venules and lay eggs. The eggs traverse through the bladder wall, and excrete in the urine. Some eggs are trapped during this process, and incite intense granulomatous inflammation resulting into nodules, polyps or masses that ulcerate, fibrose and calcify. Urinary bladder and ureter are affected early, whereas kidney and genital system are affected later. Preferentially bladder base and trigone regions are affected, resulting into bladder contraction. Initially distal ureteral dysfunction occurs which leads to ureteral dilatation. Later fibrosis results in stricture and calcification which can progress on ascending fashion towards the proximal ureter. Kidneys are not primary target of infection but are eventually affected due to ureteral obstruction and reflux.

Schistosomiasis is the most common cause of urinary bladder wall calcification in endemic regions [33]. The extent of calcification depends on the number of trapped ova in the bladder wall. Calcification begins at the bladder base in fine linear pattern, which later progresses to circumferential pattern with involvement of entire bladder wall. This circumferential urinary bladder wall calcification, resembling fetal head in pelvis on radiograph, is considered pathognomonic of chronic urinary bladder schistosomiasis [34]. The closest differential of bladder wall calcification is tuberculosis. Urinary bladder cancer is an important complication of long-standing urinary bladder schistosomiasis. Chronic bladder inflammation leads to squamous metaplasia, and

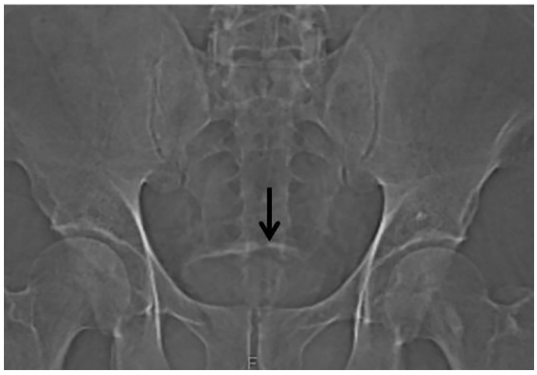


Fig. 7. Fetal head calcification of Schistosomiasis. Frontal plain radiograph of pelvic region reveals curvilinear diffuse calcification outlining the urinary bladder (black arrow).

ultimately to squamous cell cancer of urinary bladder [34]. Squamous cell cancer accounts for < 10% of bladder cancer in USA, but accounts for 75% of bladder cancer in Egypt due to endemicity of schistosomiasis [34]. It has male predominance. It is seen as focal bladder wall thickening or discrete soft tissue mass from bladder wall, usually from trigone and lateral walls. Unlike urothelial cell cancer that is mainly papillary, squamous cell cancer in bladder is sessile. Thus squamous cell cancer in bladder has muscle invasion (80%) and extensive extravesical spread [35]. Interestingly, the rate of lymph nodal and distant metastasis in schistosomiasis related bladder cancer is lower due to extensive fibrosis of draining lymphatics and capillaries. However, the prognosis is grim despite low rate of distant metastasis, due to extensive extravesical extension [32].

Plain radiographs may demonstrate linear calcifications of the urinary bladder wall and ureter. Early bladder wall calcification may have fine linear pattern near the bladder base, which later progress into extensive circumferential pattern resembling fetal head [36] (Fig. 7). Shell-like rim of calcification has no effect on bladder capacity; gradually the bladder wall becomes fibrotic and contracts, leading to capacity reduction [37]. Linear or parallel linear calcifications of ureters are initially visible in distal ureters, which may progress cranially to involve even the whole length of ureters. Voiding cystourethrogram can be beneficial to depict the vesicoureteral reflux. USG can show bladder wall calcification which appear echogenic and can also depict the wall thickening in cystitis cystica and squamous cell cancer. But the information obtained from USG is limited as compared to the cross sectional imaging.

CT is superior to other imaging modalities in demonstrating extent

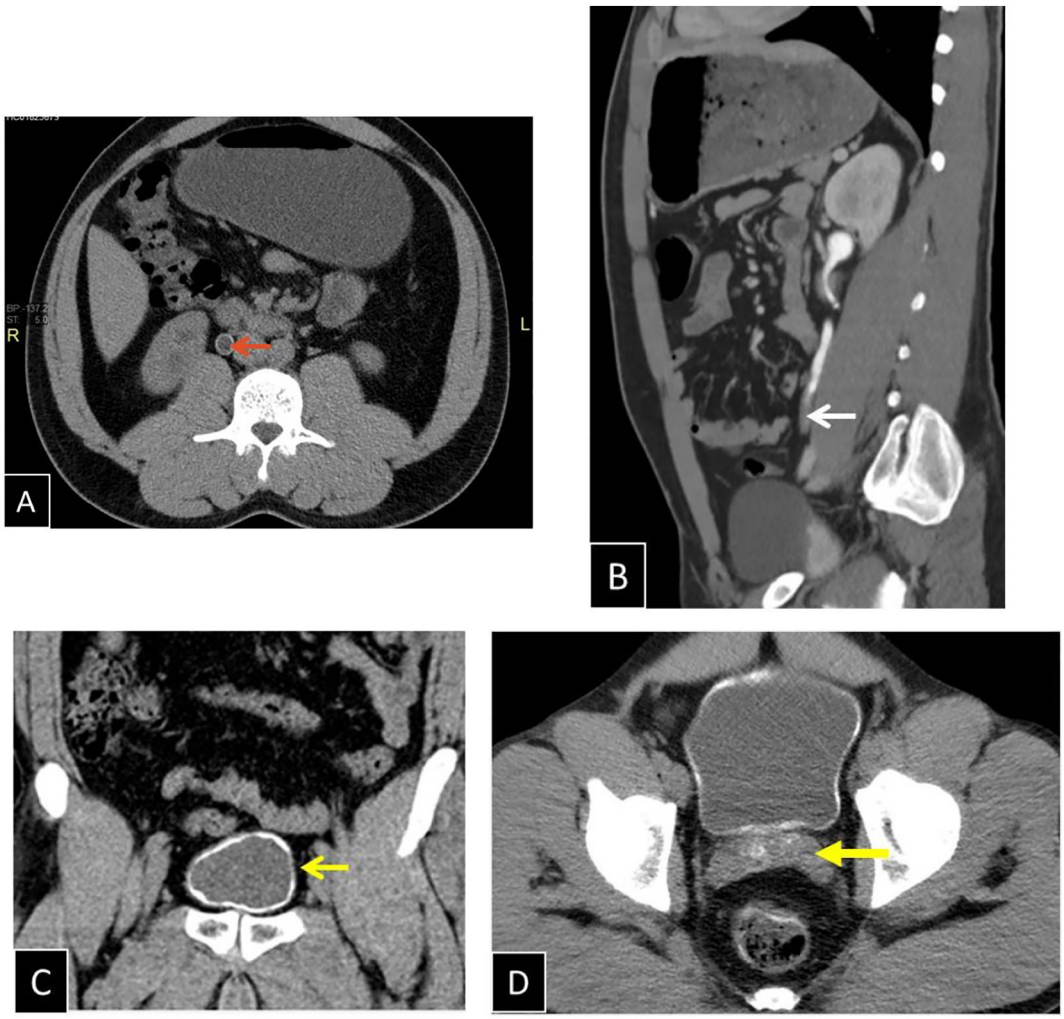


Fig. 8. CT findings in genitourinary Schistosomiasis. A) Axial non-contrast CT image demonstrates circumferential smooth mural calcification of right upper ureter (red arrow). B) Sagittal CT urography image shows mural thickening of distal ureter difficult to distinguish with malignancy (white arrow). C) Coronal non-contrast CT image showing diffuse linear “egg shell” calcification of urinary bladder (yellow arrow). D) Spotty calcifications involving prostate and seminal vesicles (yellow arrow). Also seen is diffuse calcification of urinary bladder and rectum in same image. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

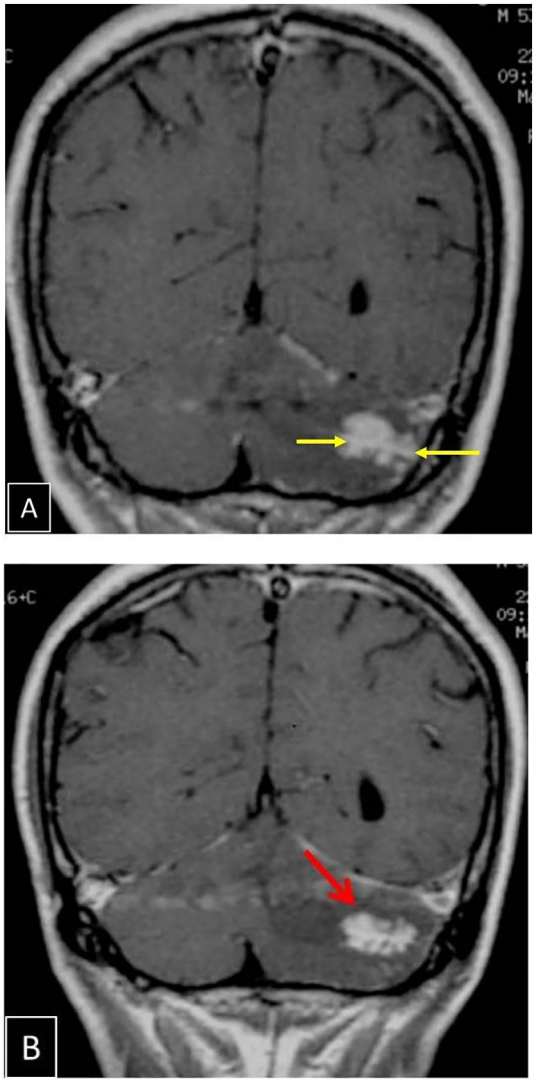


Fig. 9. Cerebellar Schistosomiasis. A, B) Coronal contrast-enhanced T1-weighted MR images show central linear enhancement (yellow arrows) surrounded by enhancing punctate nodules (red arrow) in left cerebellum. Adjacent T1 hypointensity corresponds to surrounding edema. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

of calcification associated with urogenital schistosomiasis. CT can clearly show the urinary bladder wall and ureteral calcification, even the subtle ones (Fig. 8). Even before appearance of subtle calcification, CT can show bladder wall edema and blurred fat plane between bladder wall and perivesical fat. It can also evaluate the ureteral strictures and the complications such as hydronephrosis and nephrolithiasis. CT urography can show earliest changes of altered peristalsis in the form of persistent opacification and dilatation of distal ureters. Distal ureter is affected because of the abundant blood supply [32]. Interstitial and juxtavesicular portions of ureter are frequently involved and may show mass like mural thickening [7]. Ureteral strictures, ureteritis cystica related multiple filling defects or large irregular filling defect due to squamous cell cancer can be better evaluated with CT urography. CT can depict the extravesical local spread and lymph nodal/distant metastasis of squamous cell cancer as well [34].

Genital involvement is far common in male than in female. At autopsy, Schistosoma infection of prostate and seminal vesicles has been reported in as many as half of the male cadavers where the disease is endemic [38]. Prostate and seminal vesicles are edematous initially, but

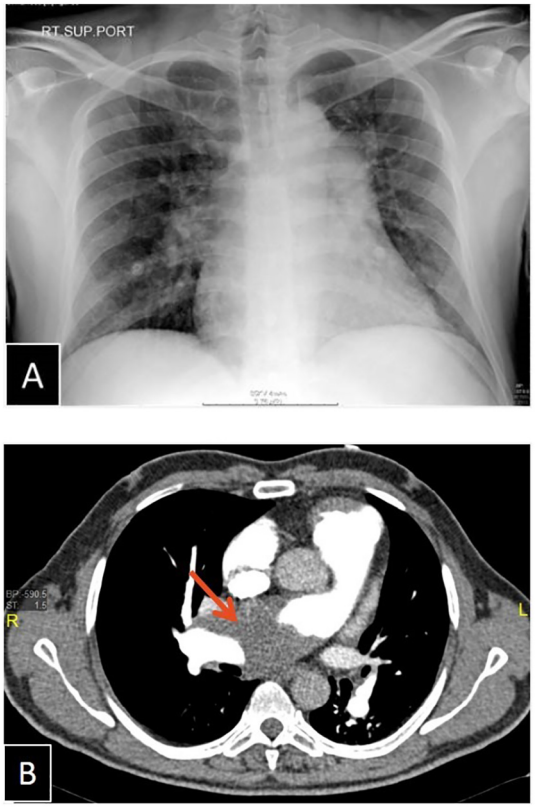


Fig. 10. Pulmonary Schistosomiasis. A) Plain radiograph of the chest shows enlarged bilateral main pulmonary arteries. B) CT chest with pulmonary angiogram shows large intraluminal thrombus in right branch of main pulmonary artery (red arrow). (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

atrophy and calcify later [39]. Calcification of prostate and seminal vesicles indicate chronic infection which shows atrophic changes and area of low signal in T1 and T2 weighted images on MRI. Testes, epididymis and spermatic cord are rarely affected [38,40]. Rarely in females vulval and perineal lesions resembling condyloma lata, tubal block and tuboovarian abscess may be seen [32].

2.7. CNS Schistosomiasis

Rarely schistosomiasis can affect the CNS. The postulated mode of spread to the CNS is through the vertebral venous plexus from abdominopelvic veins during Valsalva straining in daily routine [41]. Among different species, *S. japonicum* usually affects brain; *S. haematobium* and *S. mansoni* usually affect spinal cord. The predilection is explained by *S. japonicum* ova being smaller and devoid of the ova spine, such that the ova are not trapped caudally in venous plexus near the spine, but can reach upto the meningeal veins. To the contrary, *S. haematobium* and *S. mansoni* ova are bigger and have spines, thus their ova are trapped in spinal venous plexus, resulting into relative predilection for the spinal cord [42]. Though relatively uncommon in the literature, CNS Schistosomiasis is an important cause of epilepsy in endemic areas, affecting 1.6–4.3% of infected population [41]. Clinically it may present as headache, seizure disorder, focal neurological defect and hemiparesis, which can simulate neoplasm both clinically and on neuroimaging [43]. Diagnosis requires combination of CNS symptoms, history of exposure (freshwater in endemic area), positive serology/urine/stool samples, and typical MRI findings. CT shows single or multiple hyperdense lesions with variable enhancement surrounded by edema and mass effect. Characteristic MRI features of Schistosoma infection of brain include multiple clustered nodules with

Table 3
Summary of imaging findings in human schistosomiasis

Organ involvement	Common imaging findings
Hepatosplenic Schistosomiasis	Ultrasound findings: Periportal fibrosis, left lobe hypertrophy, mosaic liver pattern with echogenic septa outlining polygonal liver parenchyma, gall bladder wall edema. CT scan findings: "Turtle back" calcifications, periportal fibrosis, portal hypertension. MRI findings: Early periportal edema, periportal fibrosis, differentiate from liver cirrhosis, gamma gandy bodies, portal hypertension.
Gastrointestinal Schistosomiasis	CT scan findings: Characteristic tram track calcifications of colonic loops and rectum. Colonic stenosis and intestinal obstruction rarely.
Genitourinary Schistosomiasis	Plain radiographs: "Fetal head" urinary bladder calcifications, linear or parallel calcifications of ureter. Ultrasound findings: Non- specific. CT scan findings: Superior in demonstrating calcifications of urinary tract, prostate, seminal vesicles. CT urography superior in showing earliest changes of hydronephrosis, cystitis cystica or large filling defect due to malignancy.
Central nervous system Schistosomiasis	CT scan: Non-specific. MRI findings: Brain involvement with multiple clustered nodules with intense nodular and linear enhancement in arborizing pattern resembling "Buddha hand". Spinal cord expansion at distal thoracic and conus medullaris, multinodular intramedullary enhancement specific.
Pulmonary Schistosomiasis	Acute phase: Micronodules, bronchial wall thickening and ground glass densities. Chronic phase: Pulmonary hypertension, cor pulmonale.

intense nodular or linear enhancement forming unique arborizing pattern, mostly located in the cerebral white matter and basal ganglia [41] (Fig. 9). The nodules are considered to be granuloma around the ova; the linear enhancement is considered to be leptomeningeal vein obstructed by the ova [44]. Diffusion weighted imaging of lesions and proximal perifocal edema might be helpful to differentiate cerebral schistosomiasis from metastasis and glioma [45].

Spinal schistosomiasis should always be in a differential in an endemic region for non-traumatic myelopathy especially preceded by lumbar pain for hours or up to weeks [46]. Spinal cord schistosomiasis has predilection for distal thoracic spinal cord and conus region since free anastomosis exists between pelvic veins and vertebral venous plexus at these levels. It is rare for spinal Schistosomiasis to present with concurrent visceral involvement which makes diagnosis difficult [47]. MR imaging commonly shows expansion of distal thoracic cord and conus medullaris. Three patterns of contrast enhancement are described: intramedullary nodular, peripheral, and radicular [48–51]. It should be considered in the differential diagnosis of multinodular intramedullary contrast enhancement affecting the lower thoracic cord and conus medullaris in patients from endemic area. Treatment is with praziquantel and corticosteroid therapy and surgical resection in selected cases.

2.8. Pulmonary Schistosomiasis

Pulmonary involvement can be divided into acute and chronic disease. Acute pulmonary schistosomiasis is seen in nonimmune travelers following 3–8 weeks after parasite entry as a part of Katayama syndrome [52]. It results from systemic hypersensitivity reaction; eosinophils are sequestered in the lungs. Eosinophilia provides the diagnostic clue [53]. Pulmonary symptoms include dry cough, dyspnea, wheezing and chest pain. Chest radiograph shows micronodules, pleural or pericardial effusion. CT scan can show micronodules, bronchial wall thickening or less commonly macronodules, reticulo-nodular pattern, and bilateral diffuse areas of ground-glass opacities [54]. The acute phase is self-limited and subsides in few weeks without permanent lung injury [55]. Chronic pulmonary schistosomiasis is mostly seen in endemic areas, especially in hepatosplenic schistosomiasis. Portal hypertension in hepatosplenic involvement leads to portosystemic shunts; thus ova escape the portal circulation and reach the systemic circulation, thereby embolizing the pulmonary vasculature [55]. In the pulmonary vasculature, ova cause intense inflammation leading to obliterative arteriolitis, granulomatous pulmonary fibrosis, pulmonary hypertension and eventually cor pulmonale (Fig. 10).

3. Conclusion

Schistosomiasis is a common parasitic disease with multisystem involvement associated with significant morbidity and mortality. Radiologists should be aware of the typical clinical and imaging features of this entity, to include it in the differential (Table 3). Since the findings might be characteristic and even pathognomonic at instances, radiologists might be the first one in the health care provider team to suspect and point out the diagnosis. Hence, imaging appearance must always be corroborated with clinical symptoms, positive exposure, and serology or stool samples positive for schistosomiasis.

Acknowledgment

Our patients, the great source of learning.

Funding

None.

Conflict of interest

None.

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