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Case Report

Imaging findings of hepatosplenic schistosomiasis: a case report

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ABSTRACT

In our study, in a 52-year-old man, specific and nonspecific findings of Schistosoma infestation were examined using ultrasonography, computed tomography, and magnetic resonance imaging. On computed tomography, capsular and septal calcifications and contrast enhancement of the liver capsule were seen. On T1-weighted magnetic resonance images diffuse hypointensity was seen in periportal spaces; on T2-weighted images in the same spaces, diffuse hyperintensity was seen. On dynamic contrast-enhanced T1-weighted images, in these same spaces marked contrast enhancement was manifested in the late venous phase. These signal changes indicate edema due to periportal fibrotic tissue inflammation and are accepted as pathognomonic for a liver infested by Schistosoma.

Introduction

Schistosomiasis is a disease caused by parasites of the Schistosoma (S) species. *S. haematobium* causes infestation in the urinary system; *S. mansoni* and *S. japonicum* infest the colon, rectum, and liver [1]. Schistosoma infestation, in the regions of the world where it is endemic, is a major cause of liver fibrosis. Today, because of increased mobility between countries around the world, patients infected with Schistosoma may be encountered in nonendemic areas. These parasites usually live in the lumen of the intestines, whereas eggs in the mesenteric veins are carried to the liver via the portal vein, where they cause a granulomatous reaction in the periportal tissue and in the long term, cause periportal fibrosis and portal hypertension [1,2]. In schistosomiasis, the lobular parenchymal structure of the liver and hepatocytes are relatively protected and, therefore, are unlikely to develop cirrhosis, so that the liver function is usually preserved [1,2,4]. Definite diagnosis of Schistosoma infestation is based on determining the presence of parasite eggs in feces, urine, or biopsy material. Imaging modalities provide additional information to support the diagnosis [3]. Abdominal ultrasonography (US) and computed tomography (CT) can be used to evaluate specific and nonspecific findings of the disease, but these findings can also be seen in liver cirrhosis. Magnetic resonance imaging (MRI), however, in contrast to other imaging modalities, can manifest imaging findings that are accepted as pathognomonic for Schistosoma infestation in the liver [1,2,4]. This study discusses imaging

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findings in a man of North African origin with chronic hepatosplenic schistosomiasis.

Case report

A 52-year-old man infested by *Schistosoma* presented to our clinics with right upper quadrant pain, fever, nausea, vomiting, and jaundice which had increased in the prior few days. His medical history disclosed that he was an Egyptian living in Germany on business who, 4.5 years before, had undergone a cholecystectomy operation after he had received a diagnosis of Schistosoma infestation (in the region where he had lived in Egypt, *S. mansoni* infestation is widespread). Physical examination revealed icteric skin and sclera, tenderness in the right upper quadrant when palpated, splenomegaly, and fever (38.7°C). The Charcot triad was accepted as positive due to these findings. For the clinical diagnosis of splenomegaly, we considered the presence of palpable spleen whose border felt more than approximately 7-8 cm. below the costal margin. Laboratory finding abnormalities were: leucocytes, 16,400 mm^3; thrombocytes, 79,000/mm^3; peripheric eosinophilia, 8%; total bilirubin, 8.4 mg/dL; and direct bilirubin, 3.2 mg/dL. In addition, mild-to-moderate elevated liver enzymes and C-reactive protein of 58 mg/dL. No eggs were found in his feces or urine samples.

US examination showed dilatation of intrahepatic bile ducts, periportal space enlargement and increased echogenicity in periportal spaces, contour irregularities and heterogeneous echogenicity of liver parenchyma, dilatation of portal and splenic veins, and splenomegaly (largest diameter of spleen: 19 × 15 × 10 cm). Upper abdomen CT examination disclosed contour irregularities of the liver, caudate lobe hypertrophy, dilatation of periportal spaces, parenchymal retractions, capsular and septal millimetric calcifications, deepening of sulci, dilatation of the portal vein and its branches, splenomegaly, and millimetric hyperdense siderotic nodules in the spleen. On contrast-enhanced CT, contrast enhancement of the liver capsule was seen (Fig. 1A, B). Magnetic resonance imaging (MRI) demonstrated findings that had been seen in CT (except calcifications). On T1-weighted images, diffuse hypointensity was seen in periportal spaces, dilated, and elongated throughout the liver capsule. On T2-weighted images in the same spaces, linear hyperintensities were seen (Fig. 2A, B). On dynamic contrast-enhanced fat-saturated T1-weighted images, marked contrast enhancement was seen in the late venous phase in these same spaces. Also, focal dilatations of intrahepatic bile ducts, wall thickening of bile ducts, and marked contrast enhancement of bile duct walls in the late venous phase were detected and interpreted as cholangitis (Fig. 2C-E). In addition, MRI of coronal sections showed lobulated hepatic contour and parenchymal retraction due to fibrous septas (Fig. 3A). Magnetic resonance cholangiopancreatography (MRCP) examination revealed stenosis of the common bile duct due to compression from dilated portal veins, dilatation of intrahepatic bile ducts and proximal common bile duct segment, and millimetric calculi in the common bile duct (Fig. 3B). Endoscopic retrograde cholangiopancreatogram (ERCP) examination disclosed stenosis of the common bile duct, dilatation of intrahepatic and extrahepatic bile duct segments, and millimetric calculi in the common bile duct (Fig. 4). During the Endoscopic retrograde cholangiopancreatogram procedure, calculi in the common bile duct, sludge, and pus material were drained, and a stent was placed into the extrahepatic bile duct lumen for the purpose of decompression. Intravenous (IV) antibiotherapy was administered for acute cholangitis. The patient recovered, and after one week, he was discharged from hospital. One month later, an ultrasound guided true cut needle liver biopsy was performed. The biopsy material showed fibrous enlargement of periportal spaces with diffuse mononuclear cell infiltration next to normal liver parenchyma. Also, macrophages, calcified egg shells, and dark-brown pigments were seen in the periportal space. These findings strongly suggested chronic schistosomiasis.

Discussion

Hepatosplenic schistosomiasis is a chronic manifestation of *S. mansoni* and *S. japonicum* infection. The schistosomal portal

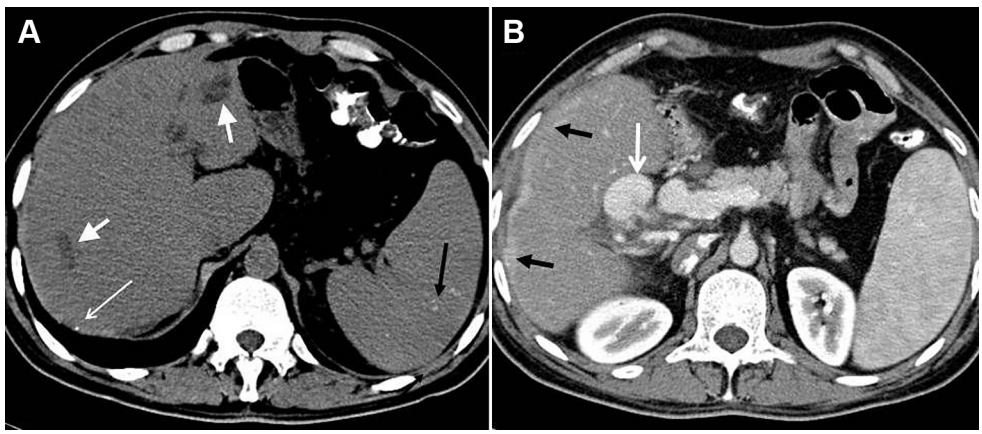

Fig. 1 – Noncontrast CT image demonstrates (A) caudate lobe hypertrophy (long arrows), intrahepatic bile duct dilatation (short white arrows), millimetric calcification in the liver capsule (open arrow), and siderotic nodules in the spleen. Contrast-enhanced, portal venous phase CT image demonstrates (B) contrast enhancement of the liver capsule (black arrows) and portal vein dilatation (white arrows).

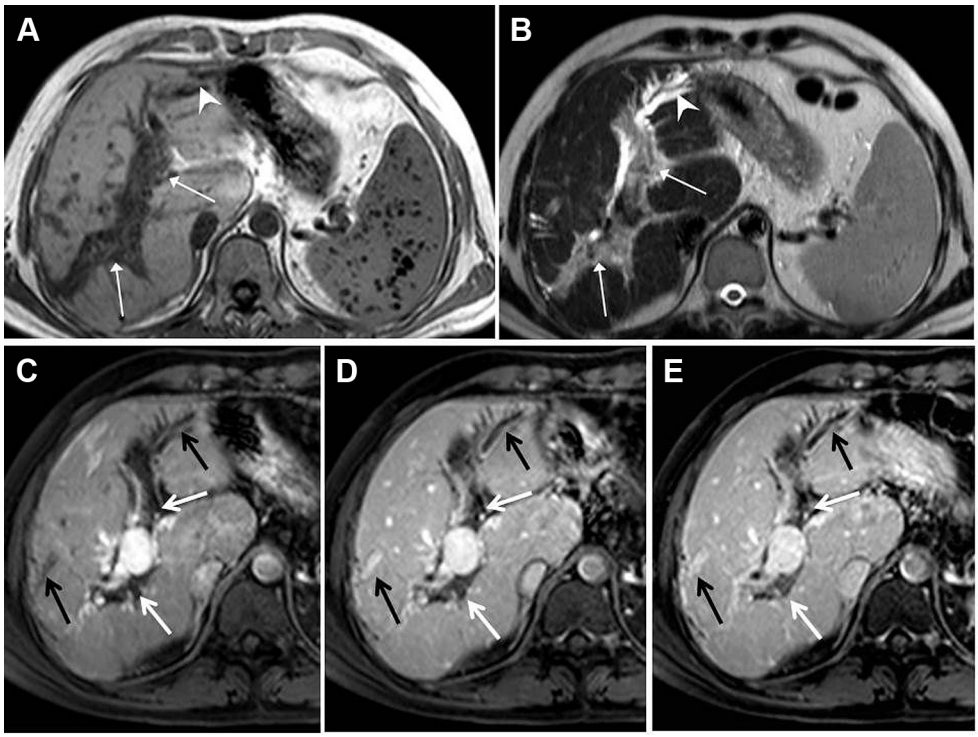

Fig. 2 – T1-W and T2-W axial MR images demonstrate widened periportal spaces elongated toward the subcapsular location at the periphery. These periportal spaces on the T1-weighted image (A) reveal a hypointense signal characteristic and, on the T2-W image (B), a hyperintense signal characteristic which reflects (the) periportal inflammatory edema due to schistosomiasis (arrows). Signal void spaces due to siderotic nodules are seen in the spleen. Dilated bile ducts are more discernible on the T2-weighted image (arrow heads). Dynamic contrast-enhanced T1-weighted fat-saturated scans at arterial phase (C), portal venous phase (D), and late venous phase (E): As time goes on, contrast enhancement increases in widened periportal spaces (white arrows). Bile duct dilatations and increased contrast enhancement on thickened bile duct walls (black arrows) compatible with cholangitis are also present.

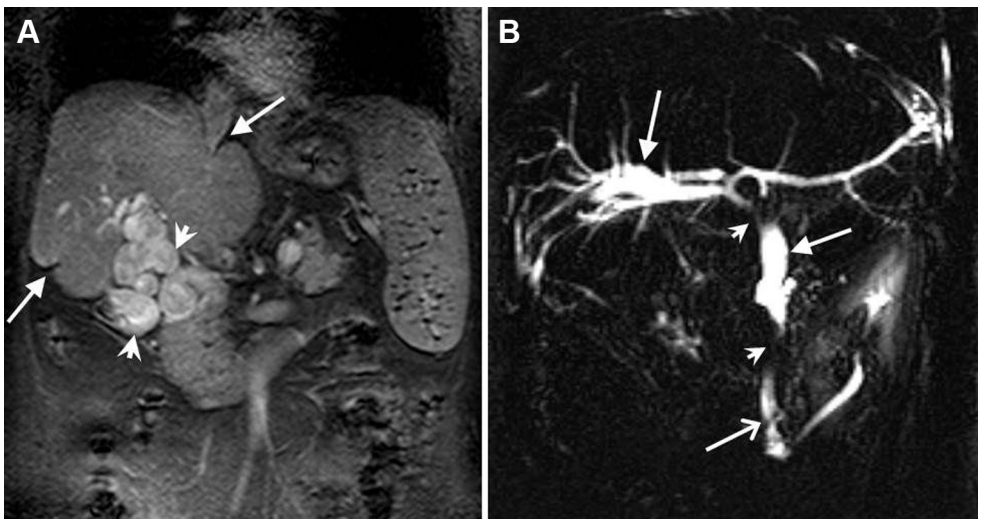

Fig. 3 – Coronal, fat-saturated contrast-enhanced T1-weighted MR image (A) demonstrates lobulated hepatic contour and parenchymal retraction due to fibrous septas (arrows) and retractions. At liver hilum, dilatation and tortuosity of the portal vein and its branches secondary to portal hypertension (arrow heads), splenomegaly, and siderotic nodules are also seen. P: pancreatic head. Magnetic resonance cholangiopancreatography scan (B) demonstrates common bile duct stenosis at the bifurcation level and distal segment due to the compression of the dilated portal vein (arrow heads), dilatation of intrahepatic and extrahepatic bile ducts (closed arrows), and microcalculi in the common bile ducts (open arrow).

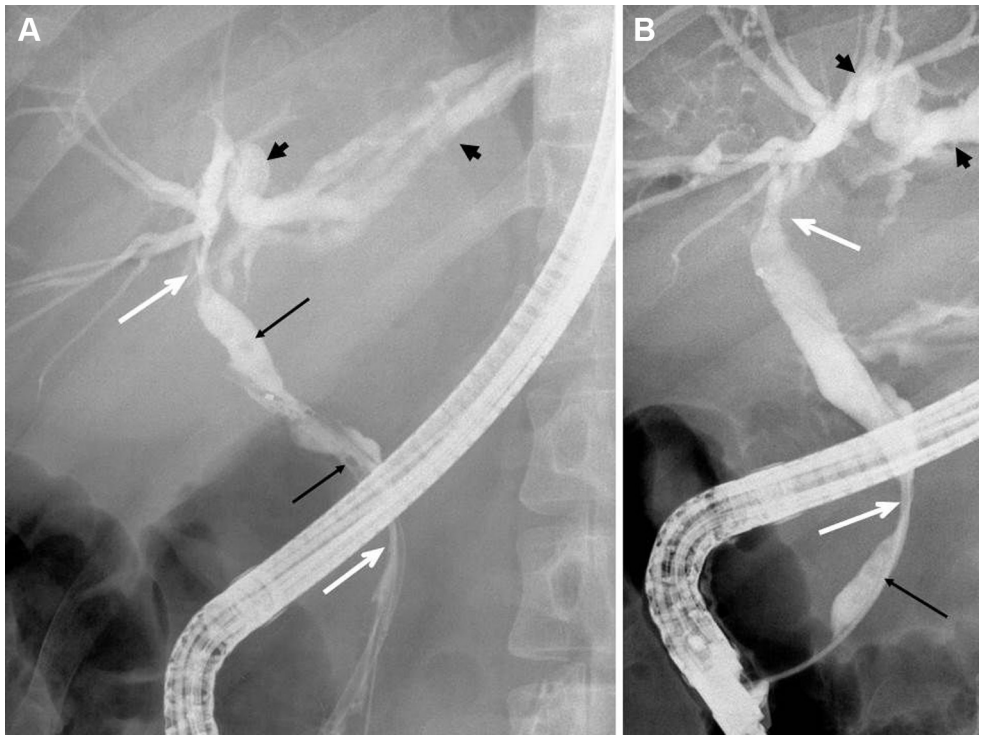

Fig. 4 – Endoscopic retrograde cholangiopancreatogram images (A, B) demonstrate common bile duct stenosis at the bifurcation level and the distal segment (white arrows), dilatation of intrahepatic and extrahepatic bile ducts (arrow heads), and millimetric calculi in the common bile duct (black arrows).

fibrosis (Symmers' pipe-stem fibrosis) as first described by Symmers (1904) is one of the most characteristic lesions of the liver [5]. It presents with an enlargement of the portal spaces by fibrosis with the portal vein branch being damaged, whereas the biliary and hepatic arterial structures remain unharmed. In contrast to other cirrhosis types, there is typically no bridging between the fibrous tracts, no nodule formation, and no injury to hepatocytes. In the active stage, portal inflammatory infiltration and accumulation of schistosome eggs in the portal areas with periovular granulomas, telangiectasia, and vascular obstruction are seen. In the late stage, a scar tissue develops around distorted, empty egg shells, and dark-brown schistosomal pigments. Microscopically, portal spaces usually become infiltrated by macrophages, plasma cells, and giant cells in response to a foreign body reaction that arises around residues of the egg shells. Patients with hepatosplenic schistosomiasis are clinically characterized by intrahepatic portal vein obstruction, portal hypertension, pancytopenia (hypersplenism), and hepatosplenomegaly. The disease usually does not show hepatocellular failure [3–5].

Abdominal US is the first imaging modality in the evaluation of patients suspected of having schistosoma infestation. US can show an irregular liver surface, a hypertrophic left liver lobe, granulomas, mosaic patterns (echogenic septas between polygonal spaces in relatively normal liver parenchyma), dilatation of the portal vein and its branches, echogenic wall thickening, splenomegaly, and siderotic nodules in the spleen [2,6]. Cross-sectional imaging modalities reflect the periportal inflammation and sequela of the fibrosis in chronic schistosomiasis [3]. CT may show calcified fibrous septa, usually vertical to the liver capsule. Capsular calcifications, irregular liver contour, and periportal fatty tissue that extends to the capsule may also be seen. Septal and capsular fibrosis with parenchymal retractions may give the liver a characteristic tortoise shell appearance. Periportal fibrosis may be shown on CT as decreased attenuation in areas surrounding the portal vein and after intravenous contrast material injection, these areas may be enhanced [3,6]. MRI is a sensitive method for the diagnosing of schistosomiasis. Except for calcifications, MRI can show all the findings of CT. Fibrous septa on T1- and T2-weighted images were seen as hypointense bands [6]. Perihepatic fatty tissue goes through into the liver along the widened fissure. Periportal fibrosis is seen as bands that follow the portal vein and can extend from the hilum to the liver capsule. On fat-saturated T1-weighted images, these bands are seen as more hypointense relative to the normal liver parenchyma. They enhance especially in the late venous phase. The most specific MRI finding in schistosomiasis is the appearance of periportal spaces: on T1-weighted images these spaces appear hypointense, and on T2-weighted images, conversely, they seem hyperintense. The signal increases as T2-weighted images reflect the periportal inflammatory edema which is pathognomonic for schistosomal infestation of the liver. This finding helps in the differentiation between inflammatory edema and periportal fibrosis.

For this reason, MRI differs from other imaging modalities by indicating early findings of schistosoma infestation and progression of the disease or regression to fibrosis after treatment. MRI shows specific findings of schistosomal infestation of the liver; therefore, MRI should be the preferred imaging modality [1,4,6,8]. MRI may also show venous dilatation due to portal hypertension, splenomegaly, and siderotic nodules in the spleen [1,7]. On MRI, these nodules, on all sequences, appear as millimetric signal void foci and do not enhance [8]. The involvement of the gall bladder is rare in schistosomiasis. In an autopsy series of 1220 specimens, gall bladder involvement accounted for only 2.5% [9].

In conclusion, in the diagnosis of the hepatosplenic schistosomiasis, MRI is the gold standard method which can show specific findings of the disease.

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