

Caso Clínico / Radiological Case Report

HEPATOBIILIARY FASCIOLIASIS*FASCIOLÍASE HEPATOBILIAR***Daniel Ramos Andrade, Luísa Andrade, Célia Antunes, Paulo Donato, Luís Curvo Semedo, Filipe Caseiro Alves**

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Abstract

Hepatobiliary fascioliasis is a parasitic disease caused by *Fasciola hepatica*, which is a trematode that primarily infects cattle and sheep, but may also affect humans in endemic areas. There are two phases of the disease: the acute one - where the parasites infect the liver parenchyma; and the subacute / chronic phase - when the parasites reach the biliary ducts and gallbladder, providing typical imaging findings. Because this disease may mimic several hepatobiliary disorders, misdiagnosis or late diagnosis is a concern. Therefore, knowledge of the typical and specific imaging findings is important in accomplishing a correct diagnosis. The authors describe a case of a 49-year-old male that presented with nonspecific liver symptoms. Liver ultrasound, computed tomography and magnetic resonance imaging showed several typical findings of the disease, which helped achieve the diagnosis.

Key-words

Parasitic Diseases; Fascioliasis; *Fasciola hepatica*; Multidetector Computed Tomography; Magnetic Resonance Imaging.

Resumo

A fasciolose hepatobiliar é uma doença parasitária causada pela *Fasciola hepatica*, um trematodo que infecta principalmente bovinos e ovinos, mas que também pode afetar seres humanos em áreas endémicas.

Existem duas fases da doença: uma aguda – em que os parasitas atingem o parênquima hepático; e uma subaguda / crónica - quando os parasitas alcançam os ductos biliares e vesícula biliar; com achados imagiológicos condizentes.

Uma vez que esta doença pode mimetizar diversas doenças hepato-biliares, podem ocorrer erros de diagnóstico ou diagnóstico tardio. Portanto, o conhecimento dos achados imagiológicos típicos e específicos é importante para se conseguir chegar ao diagnóstico correto.

Os autores descrevem um caso de um homem de 49 anos, que se apresentou com sintomas inespecíficos do foro hepático. A ecografia, tomografia computadorizada e ressonância magnética revelaram vários achados típicos da doença, o que ajudou a chegar ao diagnóstico final.

Palavras-chave

Doenças parasitárias; Fasciolíase; *Fasciola hepatica*; Tomografia computadorizada; Ressonância Magnética.

Clinical History

A 49-year-old male presented to our hospital with one month of abdominal pain, asthenia, anorexia, fever, pruritus and weight loss of 30kg. Laboratory examinations revealed eosinophilia, elevated cytolytic hepatic enzymes and slightly raised C-reactive protein.

Image Findings

An abdominal ultrasound (US) showed slight hepatomegaly and splenomegaly, with diffuse heterogeneous echotexture of the liver. Two subcapsular, hypoechogenic, ill-defined liver lesions were found, the largest one measuring about 4,3 cm (fig.1). Additionally, a tubular and serpentine hyperechogenic structure was seen inside the gallbladder lumen (fig.2).

Computed Tomography (CT) showed several hypodense micronodular lesions diffusely distributed throughout the liver, with two largest conglomerates at two subcapsular areas (fig.3). Some of the lesions were clustered in a tract-like fashion (fig.4).

Additionally, there was linear low attenuation paralleling the left portal vein branch (fig.4).

Since there was no clear diagnosis after CT and the patient's condition was still deteriorating, magnetic resonance imaging (MRI) was performed. It showed marked hyperintensity of the micronodules with a less hyperintense halo on T2w images (fig.5). On T1w sequences, the lesions were isointense to the liver parenchyma, with a peripheral hypointense halo (fig.6). Periportal tracking, evidenced by thickening and enhancement paralleling the left portal vein branch was also seen (fig.7). There was peripheral enhancement of the liver lesions (fig.8). Four small subcapsular lesions were also seen (fig.9). There was no biliary dilatation in either of the three cross sectional techniques.

According to the findings of the three imaging methods, a prospective diagnosis of ductal phase of hepatobiliary fascioliasis was made.

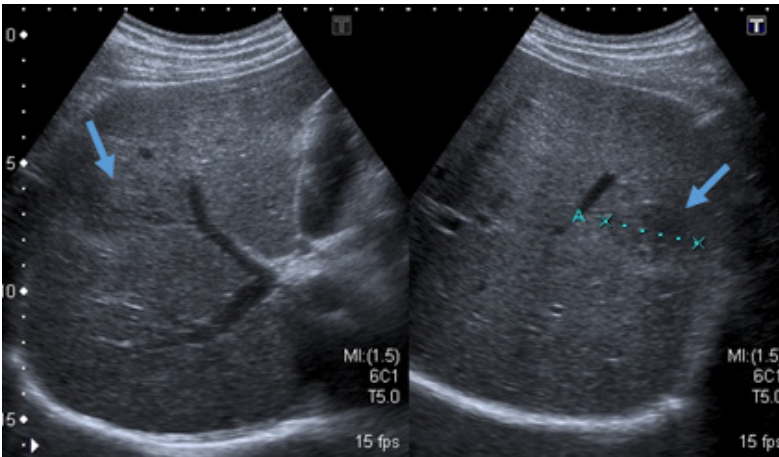


Figure 1 – Liver ultrasound – Slight hepatomegaly and diffuse heterogeneous texture of the liver; two subcapsular, hypoechoic, ill-defined liver lesions are seen (arrows).

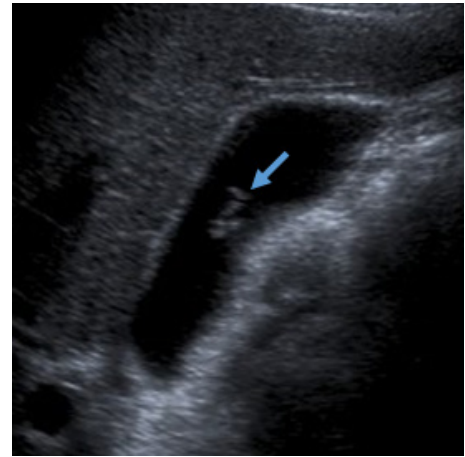


Figure 2 – Gallbladder ultrasound – A tubular and serpentine hyperechoic structure is seen inside the gallbladder lumen - a parasite (arrow).

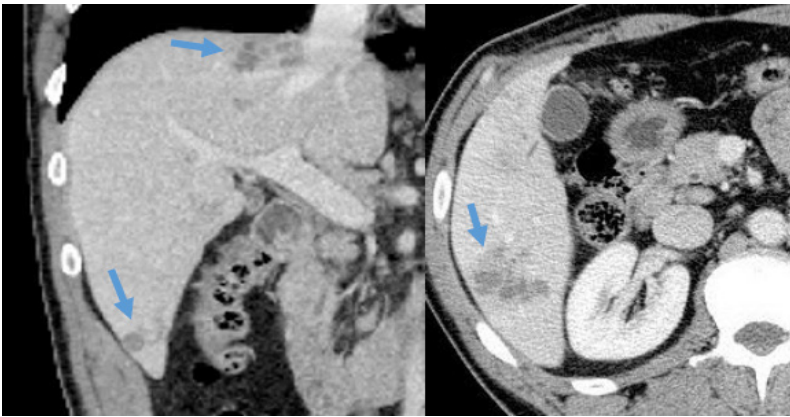


Figure 3 – Abdominal CT – Coronal reformation shows two clusters of hypodense micronodular lesions at the subcapsular areas of the VI and IV liver segments (arrows); Axial image shows a conglomerate of small round hypodense lesions at the segment VI of the liver (arrow).

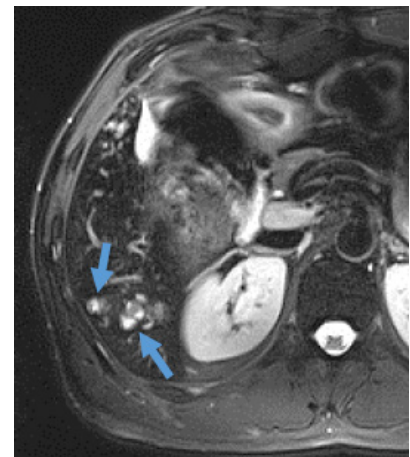


Figure 5 – MRI (T2wFS) – Hyperintense small nodular lesions, surrounded by a thin halo of less hyperintense tissue, at the segment VI of the liver (arrows).

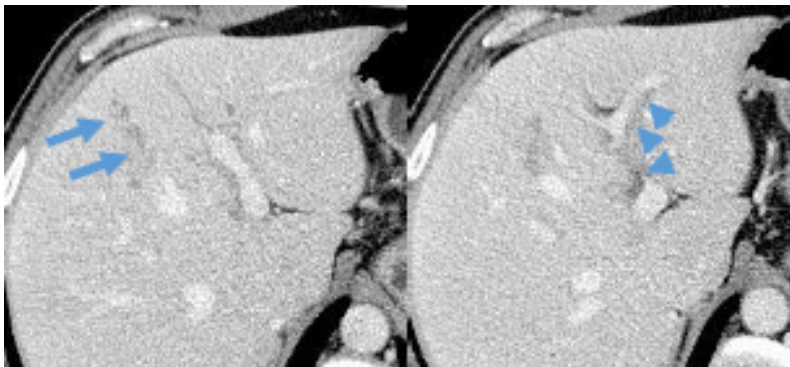


Figure 4 – Abdominal CT – In the segment IV of the liver the small hypodense lesions are distributed in a tract-like fashion (arrows); Linear low attenuation parallel to intrahepatic branches of the portal vein and left portal vein branch - periportal tracking (arrowheads).

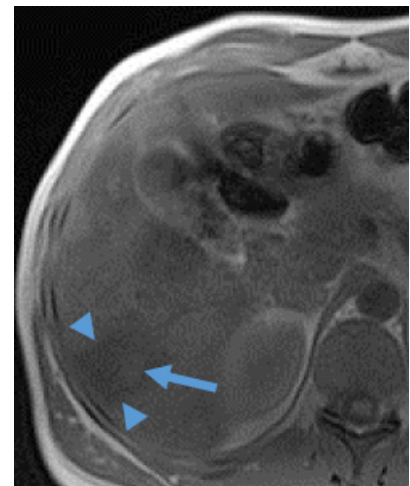


Figure 6 – MRI (T1w) – Isointense nodular lesion (arrow) surrounded by hypointense liver parenchyma - edema (arrowheads).

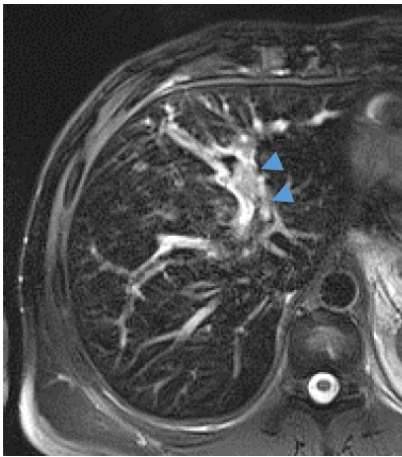


Figure 7 – MRI (T2wFS) - Hyperintense thickening of the periportal tissue near the left portal vein branch - periportal tracking (arrowheads).

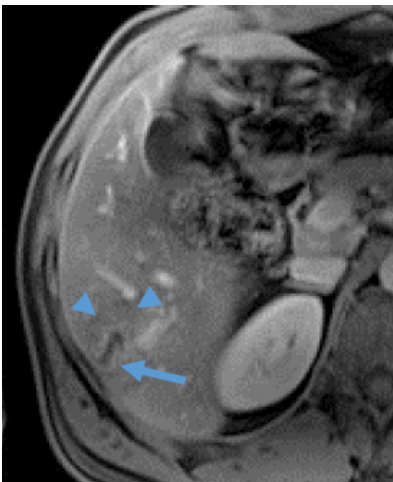


Figure 8 – MRI (T1w after ev gadolinium) – Peripheral contrast enhancement of the lesion (arrow), with a surrounding parenchymal area of less contrast enhancement - edema (arrowheads).

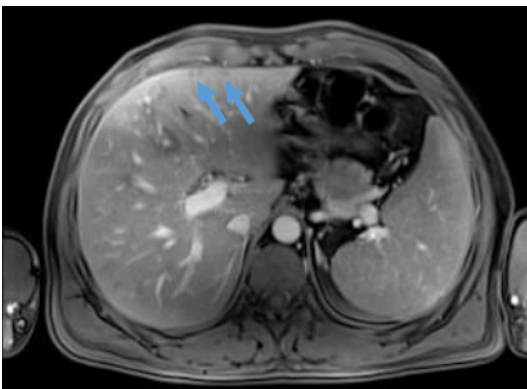


Figure 9 – MRI (T1w after ev gadolinium) – 4 small hypovascular micronodular areas at the anterior margin of the left lobe, at the site where the parasites perforated the liver capsule (arrows).

Discussion

Hepatobiliary fascioliasis is a parasitosis caused by *Fasciola hepatica*, which is a trematode that infects mainly cattle and sheep, but may also occur in humans who inadvertently ingest water plants with metacercaria¹. Human infection is found worldwide in areas where herbivores are raised, mostly in developing countries, but also in Western Europe and Australia². While some authors believe the disease is rare in Portugal, others describe the country as an endemic zone, particularly the northern (rural) areas³. The ingested metacercaria excyst in the duodenum, penetrate the bowel wall and migrate through the peritoneal cavity

and via the Glisson's capsule into the liver, where the larvae slowly migrate through the parenchyma, leaving multiple small cavities, with inflammation, hemorrhage and necrosis in their paths^{1,2}. This parenchymal phase may last two to several months. When the larvae reach a large bile duct they mature into adult flukes and start to lay eggs. In this ductal phase, the adult parasites may reach up to 40mm in length and 13 mm in width and can remain in the bile ducts or gallbladder for a decade¹.

Common symptoms of the disease are right upper quadrant pain, fever, weight loss, fatigue, pruritus, dyspepsia and vomiting⁴. Bloodwork typically shows inflammatory changes, with eosinophilia and abnormal liver function tests¹.

Diagnostic confirmation is usually achieved with serology and/or visualization of eggs in aspirated bile, liver tissue or stool^{1,5}. Treatment with an antiparasitic is usually efficacious. Because this disease may mimic several hepatobiliary disorders and because of the fact some patients may remain asymptomatic, misdiagnosis or late diagnosis is very frequent. The differential diagnosis includes ascariasis, hydatidosis, amebiasis and pyogenic microabscesses. Therefore, knowledge of the typical and specific imaging findings is important in achieving a correct diagnosis^{5,6}.

In the parenchymal phase, US findings are nonspecific and include multiple focal hypoechoic or anechoic nodules or diffuse involvement of the liver (heterogeneous echotexture), which correspond to the cavities the parasites leave behind, that are filled with blood, pus or necrosis^{1,6}. CT typically demonstrates multiple, hypodense, tract-like or nodular lesions. These agglomerates of microabscesses are first found in the subcapsular areas and show slow evolution on follow-up examinations^{1,2,6}. These lesions are indistinguishable from microabscesses of other etiologies, although they do not coalesce to form large abscesses, unlike pyogenic abscesses¹. MRI may show ill-defined hyperintense lesions on T2w images and hypointense lesions on T1w images, that may show peripheral contrast enhancement, which correlates with a halo of mononuclear and eosinophilic infiltration surrounding the parasitic cavities¹. Capsular thickening, enhancement and hyperintensity on T2w sequences may be seen in the sites where the parasites penetrated^{1,6}. Periportal or peridiaphragmatic enlarged lymph nodes and perihepatic fat stranding may also be seen in either of the imaging methods⁶. Subcapsular hematoma is a rare complication that can occur between the two phases due to the migration of flukes from the liver parenchyma to the bile ducts¹.

In the ductal phase, US and CT typically show intra-hepatic or common bile duct dilatation and tortuousness, due to the intraluminal parasites or the hemorrhage / inflammatory reaction they incite, and may even show adult flukes in the dilated bile ducts or gallbladder. US is capable of documenting the flukes moving inside the ducts or gallbladder^{1,6}. Periportal tracking, evidenced by low attenuation / T2w hyperintense tracks along the portal triads, may also be seen at CT / MR respectively¹. MRCP is also able to demonstrate bile duct dilatation and tiny filling defects caused by the flukes themselves, that simple MR or CT may fail to recognize^{1,2}. The parenchymal findings may regress or may coexist during this phase.

The imaging findings of our patient of multinodular hypoechoic / hypodense / T2w hyperintense lesions and periportal tracking are in accordance with several other case reports⁶. Although there was no biliary dilatation and there were still microabscesses typical of the parenchymal phase,

evidence of parasites inside the gallbladder immediately allocates the disease in its ductal phase. This diagnosis was suggested after MR and it was confirmed through blood serologies. The patient was treated with oral triclabendazole and improved dramatically.

In conclusion, the hypothesis of hepatobiliary fascioliasis should be raised in an appropriate clinical setting, in patients from endemic areas, when compatible imaging findings are found^{5,6}.

References

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