Uncommon Parasitic Infection: Fascioliasis.

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Learning objectives

Review radiological findings by ultrasound and CT scan of the Fasciola hepatica infection. Show key points for the early diagnosis, detection of possible complications, and differential diagnosis.
Background

INTRODUCTION

The fascioliasis is a zoonosis caused by a parasitic trematodes Fasciola hepatica and called f. Gigantica. Normally infect the herbivorous animals, man is an intermediate host that is infected by ingestion of water or certain contaminated food (usually aquatic plants such as water cress). The WHO considers it a major parasitic disease, because it affects South America, Oceania, Africa, Asia and Europe. It is a very prevalent disease in developing countries and within Europe can be found in Spain, Portugal, Italy and France, where they are endemic, with a prevalence of 3 %.

The highest prevalence is found in Bolivia (72% of the faeces analyzed contain eggs), remain in these hyperendemic areas more commonly in women. Treatment is with anthelmintic drugs, such as albendazole with excellent response. (Figure 6 and 7).

Symptoms are nonspecific, hence the importance of knowing their features in the image tests.
LIFE CYCLE OF THE HEPATOBILIARY FASCIOLIASIS

Fasciola is a parasite plane, spindle-shaped (Figure 1). It is hermaphroditic and to complete their life cycle requires two guests: one intermediate that are the freshwater snail (Lymnaea: figure 3) and another final, which can be several herbivorous animals, including humans (Figure 2).

In the definitive host, the parasite lives in the bile ducts from where produces eggs that are eliminated with the feces to the external environment. Once contact with the water hatch and a ciliated miraciclo seeks and infects the intermediate host, a freshwater snail. Inside the snail are reproduced cercariae, which are released and are anchored in the form of metacercariae (cysts with larvae) on aquatic plants, such as watercress, water lettuce or parsley.

It is after the ingestion of these plants when they leave the metacercarial cyst in the gastric lumen, penetrating the wall of the small intestine and turning to the peritoneum, where they migrate to the liver.

Then cross the Glisson capsule (PARENCHYMAL PHASE), where digest hepatocytes and advance looking for the bile ducts. This is where the larva hosts and develops up to be adults and begin to produce eggs (DUCTAL PHASE), closing the cycle. Within the liver migrates to the bile ducts, causing multiple cavities with hemorrhage, necrosis, fibrosis, infection or even abscesses.

The adult Fasciola hepatica can live up to 10 years in humans, being the interval between the infection and the production of eggs for approximately three to four months.

The young parasites few millimetres measured while the adults come to 20-40 mm in length and 8-13 mm wide.

The pathologic analysis of the liver shows parasitic granulomas with necrosis surrounded by abundant eosinophils and mononuclear cells, with fibrosis of the liver tissue adjacent. The calcifications are rare, and can be found in cases with chronic disease.

Diagnosis is based on the clinical, laboratory tests and imaging.

DIAGNOSIS

Is based on the clinical, laboratory tests and imaging.
Clinical diagnosis:

It is difficult because the symptoms are very nonspecific: fever, vomiting, hepatomegaly, abdominal pain, hives, ascites and anorexia in the parenchymal phase; diarrhea and cramps by bile formation of stones in the ductal phase.

Laboratory tests:

- Direct:

  1. Analysis of feces, which is negative until the parasite reaches the bile ducts and begins to remove eggs, to the approximately 10 weeks.

  The trematodes adults and eggs can be found in duodenal fluid aspirate or in the gallbladder after cholecystectomy


  - Indirect immunological techniques: serology, detecting antigens in serum, intradermal and in feces by ELISA (enzymelinked immunosorbent assay (ELISA). In serum is detected at 2 weeks of the infection, which may monitor the response to treatment with the serum levels of immunoglobulin G.

Imaging tests

Include ultrasound, computed tomography (CT) and magnetic resonance imaging (MRI). Their role is important to help the initial diagnosis and to distinguish these lesions from other focal liver.

• ULTRASONOGRAPHY

Parenchymal Phase

The findings at this early stage, in the first 8 weeks after the infection are nonspecific. Include focal hypoechoic lesions, hyperechoic or diffuse liver involvement. In mild infections can be observed a diffuse increase in the hepatic echogenicity while in severe infections irregular hypoechoic lesions throughout the parenchyma.

Ductal Phase
Generally parenchymal phase is resolved when starts the ductal one, although it is possible to see injury hyperechoic coexisting with the start of the ductal in some cases.

Once at 8 weeks of infection occurs parietal dilation and thickening of the bile ducts. At the beginning are shown as thin hypoechoic lines parallel to hyperechogenic portals vessels. After 12 weeks the biliary ducts dilate and become tortuous, still possible sometimes observe the parasites moving in the pipeline or the gallbladder.

• COMPUTED AXIAL TOMOGRAPHY

Is the most used technique for the diagnosis of fascioliasis.

Parenchymal Phase

The findings in this phase include subcapsular hypodense round, small and grouped lesions, with morphology and serpiginous peripheral contrast enhancement. In the first few weeks after the infection these lesions are subcapsular, progressing toward the sixth week to morphology tortuous and grouped to be moving toward the central part of the liver, showing the "sign of tunnels and caves" (Figures 4 and 5). You can be thickening and enhancement of the liver capsule secondary to the penetration of the parasites by the same, and sometimes even sees the path of migration of the parasite by the liver.

Ductal phase

At 8 weeks of the infection causes thickening and dilation of biliary ducts parallel to the portal vessels and the parenchymal lesions go away quickly. The periportal areas are displayed progressively hipoatenuadas until week 10, it is indistinguishable dilation ductal. In the hepatic parenchyma residual calcifications can be seen in the chronic phase, although uncommon. Sometimes the parasites can be seen in the bile.

• MRI

Parenchymal Phase

In T2 weighted sequences it can be seen the entry areas of the parasite as capsular hyperintense areas. The migration routes are shown as lines hypointense on T1 and hyperintense in T2. The parenchymal lesions have leathery are hyperintense on T2 and hypo in T1, with peripheral enhancement after administering intravenous gadolinium.

Phase Ductal
Overage ductal dilatation of the common bile duct is little appreciated in MRI, as lines hyperintense on T2 parallel to other hypointense that correspond to the portal vessels. There are irregular fibrotic scars and heterogeneous capsular and subcapsular. Repletion defects of linear intermediate signal, corresponding to the worms can be seen in the dilated duct.

**COMPLICATIONS OF THE FASCIOLA HEPATICA INFECTION**

There may be different localizations of the parasite during the migration by the peritoneum, or after the step into the portal system from the liver. As well, atypical locations are the pancreas, spleen, kidneys, intestine and more rarely the spinal cord. However the most common complications are cholangitis, cholecystitis, subcapsular hematoma and hemorrhage.

**Acute cholecystitis and cholangitis**

Stage chronic fascioliasis is characterized by recurrent episodes of biliary colic and cholecystitis. Acute cholangitis with abscess formation may show nodular lesions hipoatenuadas that correspond to dilated bile ducts that are located centrally. Abscesses can be multifocal, showing how fluid collections with hypodense capsula that simulates a pyogenic abscess. The content may also be hyperdense by be purulent or contain hemorrhage. It is surrounded by a halo hypodense by edema, presenting relace with minimum contrast.

Unlike the pyogenic abscess, microabscesses by Fasciola hepatica do not combine to form an abscess more, do not tend to increase in size and progress very slowly. An abscess organized you can simulate a granuloma necrotic. This pathology should be included in the differential diagnosis of liver abscesses organized in endemic areas.

**Subcapsular hematoma and hemorrhage**

Subcapsular hematoma is a rare complication of the migration of the parasite during hepatic phase. If associated with eosinophilia should suggest the diagnosis of polyarteritis nodosa or invasive distomatosis. Rarely occurs a massive bleeding that put at risk the life of the patient. The injuries can be reduced and disappear or extend toward the center of the liver when the parasite migrates to the bile ducts of greater size.

**DIFFERENTIAL DIAGNOSIS**

The diagnosis is difficult because of the broad spectrum of clinical manifestations, still must be a high index of suspicion. The urticaria, eosinophilia and hepatic lesions
are the major clinical signs and symptoms that are common with other infections. We must discard cholecystitis, hepatitis, cholangitis, brucellosis, liver abscesses and liver carcinoma primary or secondary. It is common to delay the diagnosis, leading to unnecessary procedures, such as colangiopancreatograftias endoscopic retrograde (ERCP), cholecystectomy or segmentectomy liver. The delay in treatment increases the risk of formation of pigmented biliary lithiasis and makes it necessary to use more aggressive techniques, such as percutaneous drainage of abscesses, why it is vital the image tests for the differential diagnosis.

We primarily to distinguish it from pyogenic abscess, and amebic of hydatid cysts.

Amebic liver abscess

Is caused by Entamoeba histolytica which is acquired by ingestion of food or water contaminated with the protozoa. The abscess may rupture the peritoneum and cause generalized peritonitis, or break the biliary tract or pericardial or pleural space. Mortality from this disease is mainly due to the complicated liver abscess

In ultrasound shows how hypoechoic lesion, oval or round dome near the liver, with internal echoes forming levels and without a well defined wall. In TC is manifested as round lesions with well-defined attenuation values in the range of the liquid. It is characteristic that a thin capsule and enhances contrast surrounded by edema.

Another important finding is the presence of extrahepatic manifestations, such as allocation of the chest wall, pericardial, pleural cavity and adjacent organs by the extension of amebic abscesses. In RM have low signal sequences in homogeneous potentiated in T1 and high in T2.

Hydatid cysts

Are caused by Echinococcus granulosus, a parasite endemic in geographic areas where live sheep throughout the world. The liver is the main hosting these parasites, being the image of the tests to diagnose this disease in most cases. The serological tests are used to confirm the disease or as a screening population.

In the ultrasound findings vary with the type of cyst, and it may also be submitted as unilocular cyst, with multiple layers in your wall, contain membranes, vesicles, be heterogeneous, with sand or have calcified hydatid his wall. When it is clear, a hydatid membrane internal notes is the sign of the "water lily", by its morphology within the cyst that reminds this flower.

In TC shows hypodense and well defined lesion with visible wall, calcified in half of the cases. In MRI the cysts are markedly hypointense in T1 and hyperintense in T2. The
vesicles are present when daughters are relatively hypointense compared to the matrix that surrounds him in both T1 and T2.

**Pyogenic liver abscesses**

Visceral abscess is the most frequent and could cause death in up to 14% of the cases. It usually starts by hematogenous spread or bile of germs, usually gastrointestinal. The serologic tests tend to be negative, so we have to suspect this entity when not get positive serology to parasites. The microabscesos pyogenic diseminas multiple lesions are similar to microabscesses fungal for immunocompromised patients. Can simulate fascioliasis, distinguishing itself from this in that do not affect the subcapsular area.

In ultrasound may be submitted as nodules or hypoechoic areas of parenchymal distortion poorly defined.

In TC are displayed as multiple injuries small well-defined.

In MRI the signal of the lesions varies depending on its protein content, but are usually hypointense signal intensity on T1W and hyperintense in T2W. After administering iv contrast, as in the TC, appears a ring of enhancement. In occasions may have intralesional air.
Images for this section:

**Fig. 1:** Specimen of *Fasciola hepatica* with its flattened morphology and leaf shape.  
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Fig. 2: Life cycle of the Fasciola hepatica.

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Fig. 3: Specimen of aquatic snail that is intermediate host of the parasite Fasciola hepatica.

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Fig. 4: Axial abdominal CT scan with oral and intravenous contrast in portal phase. Multiple hypodense rounded images grouped and linear serpinginosas predominance of subcapsular in segments 5 and 6 of the Couinaud classification. Extend toward the central region and enhance liver in the periphery after contrast, images representing the sign of the tunnel and cave, formed by sterile tissue necrosis by digestion of the parasite, typical of the parenchymal phase of Fasciola hepatica infection.

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Fig. 5: Coronal Reconstruction of the same patient as in figure 4.

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**Fig. 6:** Axial abdominal CT scan with oral and intravenous contrast in portal phase, where they are checked the disappearance of the lesions after the etiological treatment.

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Fig. 7: Coronal Reconstruction of the same patient in figure 6, with resolution of the lesions.

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Conclusion

The role of ultrasound in the parenchymal phase is limited, being more useful in ductal phase. CT and MRI are useful to demonstrate the evolutionary stage of infection.

It is important become familiar with radiographic signs that, in combination with clinical suspicion and serology, allow you to reach an early diagnosis, demonstrating the exact stage of the disease, enabling appropriate treatment and prevent long-term complications.
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