Inflammatory-infectious disease of the liver. Differential diagnosis and radiologic-clinic correlation.

<table>
<thead>
<tr>
<th>Poster No.</th>
<th>C-0077</th>
</tr>
</thead>
<tbody>
<tr>
<td>Congress</td>
<td>ECR 2010</td>
</tr>
<tr>
<td>Type</td>
<td>Educational Exhibit</td>
</tr>
<tr>
<td>Topic</td>
<td>Abdominal Viscera (Solid Organs)</td>
</tr>
<tr>
<td>Authors</td>
<td>G. Aguilar Sánchez, J. Sánchez Parrilla, A. Radosevic, N. Risueño Pedrosa, I. Fuertes Vallcorba, M. Busto Barrera; Barcelona/ES</td>
</tr>
<tr>
<td>Keywords</td>
<td>Inflammatory-infectious liver disease, differential diagnosis, radiologic-clinic correlation</td>
</tr>
<tr>
<td>DOI</td>
<td>10.1594/ecr2010/C-0077</td>
</tr>
</tbody>
</table>

Any information contained in this pdf file is automatically generated from digital material submitted to EPOS by third parties in the form of scientific presentations. References to any names, marks, products, or services of third parties or hypertext links to third-party sites or information are provided solely as a convenience to you and do not in any way constitute or imply ECR's endorsement, sponsorship or recommendation of the third party, information, product or service. ECR is not responsible for the content of these pages and does not make any representations regarding the content or accuracy of material in this file.

As per copyright regulations, any unauthorised use of the material or parts thereof as well as commercial reproduction or multiple distribution by any traditional or electronically based reproduction/publication method is strictly prohibited.

You agree to defend, indemnify, and hold ECR harmless from and against any and all claims, damages, costs, and expenses, including attorneys' fees, arising from or related to your use of these pages.

Please note: Links to movies, ppt slideshows and any other multimedia files are not available in the pdf version of presentations.

www.myESR.org
Learning objectives

- Recognize and describe the radiological appearance of the inflammatory-infectious processes (common and uncommon) that may involve the liver, relating them to the clinical setting.
- Assess the role of radiologist and different imaging modality in the detection, characterization, and management of patients with inflammatory-infectious liver disease.

Background

The **six common pathogenetic mechanisms** by which bacteria can form liver abscesses are ascending biliary infection, portal bacteremia, septicaemia, direct extension from intraperitoneal infection, direct trauma to the liver, and secondary infection of metastatic cancer.

Infectious liver diseases can be accurately evaluated with ultrasonography (US), computed tomography (CT), and magnetic resonance (MR) imaging.

Characteristic changes in US echogenicity, CT attenuation, or MR imaging signal intensity and typical enhancement patterns can contribute to the diagnosis of specific infectious diseases, including abscesses, parasitic diseases, fungal diseases, granulomatous diseases, viral hepatitis, and other less common infections.

In most cases, guided needle aspiration or biopsy is needed for identify the type of infection. In addition, epidemiological and clinical information remains extremely important for obtaining a more accurate presumptive diagnosis.

In hepatic abscess, early diagnosis and imaging-guided percutaneous drainage have markedly reduced both the mortality rates and the need for surgery.

Imaging findings OR Procedure details
The wide variety of inflammatory hepatic diseases evaluated correspond to toxic acute hepatitis, acute viral hepatitis, acute alcoholic hepatitis, chronic hepatitis, pyogenic abscess, echinococcal disease (hydatid cyst), amoebic liver abscess, brucellar hepatic abscess, fungal diseases, uncommon hepatic infections in AIDS patients (tuberculous hepatic abscesses and *Mycobacterium avium complex* (MAC) infections), septic cholangitis with multiple hepatic microabscesses/abscesses caused by different pathogens (*Cytomegalovirus, Cryptoporridium*), infected hepatocarcinoma and infected hepatic metastases.

We show hepatic abscesses secondary to inflammatory-infectious abdominal processes (acute pancreatitis, diverticulitis, appendicitis, inflammatory bowel disease), surgery of the gastrointestinal tract or pelvic organs, blunt trauma, chronic alcoholism, corticosteroid therapy, and secondary to silent underlying malignancy (colorectal neoplasm).

In all cases ultrasonography and CT was performed including guided aspiration or biopsy for diagnosis, realizing in some cases MR, and in many cases percutaneous drainage (guided by US or CT) as a therapeutic procedure.

**PYOGENIC LIVER ABSCESS**

Pyogenic liver abscesses are focal areas of infection within the hepatic parenchyma. These abscesses may be single or multiple and result from liver invasion by a variety of bacteria.

Generally pyogenic liver abscesses are caused by gram-negative enteric bacteria, especially *Escherichia coli, Klebsiella pneumoniae, Streptococcus faecalis* and *Proteus vulgaris* being the major contributors. In other cases the infection is mixed and almost always associated with resistant staphylococcus. The role of anaerobic organisms, especially Bacteroides and Clostridium, has been increasingly recognized as culture techniques have improved. Infection by multiple organisms is frequent.

The six common pathogenetic mechanisms by which bacteria can form liver abscesses are ascending biliary infection, portal bacteremia, septicaemia, direct extension from intraperitoneal infection, direct trauma to the liver, and secondary infection of metastatic cancer.

- **Septic cholangitis**

Septic cholangitis secondary to cholelithiasis, neoplasms or biliary tract stenosis may produce multiple hepatic abscesses.

Bacteria cause most cases of infectious cholangitis. Viral cholangitis primarily affect immunocompromised patients.
**Bacterial acute cholangitis (BAC)** is a potentially life-threatening disease, usually in the setting of obstruction. The Charcot clinical triad includes fever, pain, and jaundice occurring in fewer than 75% of patients. Development of bacterial acute cholangitis requires biliary contamination, stagnant bile, and increased intrabiliary pressure. Obstruction of the common bile duct (CBD) by stones is still the most frequent cause. Biliary cultures are polymicrobial in 30%-80% of patients; gram-negative rods are found in 88% of these cases.

Acute complications of bacterial acute cholangitis include sepsis, hepatic abscesses, portal vein thrombosis, and bile peritonitis. Hepatic abscesses can be clinically silent and detected only with imaging.

Dilatation of intrahepatic biliary ducts occurs in all cases affecting central, diffuse or segmentary ducts. There is a diffuse and concentric wall thickening and enhancement of the common bile duct in the majority of patients, associated with enhancement and thickening of intrahepatic biliary duct walls. Pneumobilia can also be present in cases of BAC. Parenchymal changes are likely related to extension of the inflammatory process into the periportal tissues and surrounding liver. Patterns of enhancement can be wedge-shaped (most frequent), peripheral patchy, or peribiliary in distribution.

Antibiotic therapy alone is inadequate for treatment and is associated with high mortality rates. Either endoscopic or percutaneous biliary drainage is necessary to decompress the biliary tree and thus minimize bacterial and endotoxin spillage into the bloodstream.

**Recurrent pyogenic cholangitis (RPC)** is a progressive biliary disease characterized by recurrent episodes of bacterial cholangitis. It is associated with biliary tract ectasia, focal strictures, and formation of intrahepatic pigment stones. Abscesses are encountered in 20% of cases. They can be distinguished from bilomas owing to the presence of an enhancing rim.

**Acquired immunodeficiency syndrome (AIDS) cholangiopathy (HIV-related cholangitis)** is a form of secondary sclerosing cholangitis that affects patients who are severely immunocompromised, with a CD4 count less than 100/mm3. The liver and biliary trees are targets of infections in human immunodeficiency virus (HIV) positive patients.

The clinical findings are nonspecific but often include right upper quadrant pain and abnormal results on liver function tests. The latter are commonly observed in HIV patients and can be multifactorial, ranging from drug reactions to hepatitis. Therefore, imaging plays a crucial role in assessing HIV patients suspected to have liver or biliary infections. The bile duct and gallbladder walls are usually thickened.

Diagnosis is based on duodenal or papillary biopsy, with the results showing biliary inflammatory changes and the associated pathogens. In 50% of cases, no definite
Any inflammatory/infectious intraabdominal focus may produce a portal venous dissemination causing a hepatic abscess.

Conditions predisposing to anaerobic liver infection include inflammatory bowel disease, malignancy and/or surgery of the gastrointestinal tract or pelvic organs, biliary tract disease, blunt trauma, diabetes mellitus, chronic alcoholism, and corticosteroid therapy. (Figure 7) (Figure 8) (Figure 9) (Figure 10)

Solitary hepatic abscesses, particularly in community hospitals, are often cryptogenic without clear predisposing cause.

The clinical presentation of hepatic abscesses is quite variable. Pyogenic liver abscess is frequently an indolent condition without signs or symptoms suggestive of an acute hepatic or right upper quadrant infection.

A bacterial liver abscess may be suspected when a patient develops fever, leukocytosis and right upper quadrant abdominal pain. Often, especially in elderly patients, the presentation is subtle and involves an indolent process with loss of appetite, intermittent or low grade fever, and dull abdominal pain.

Patients may have clinically occult abscesses that present with only weight loss and vague abdominal pain. At the other end of the clinical spectrum, patients may present with profound septicaemia.

(Figures 11-12) (Figures 13-14)

The sonographic appearance of pyogenic abscesses runs the gamut from hypoechoic to hyperechoic lesions with varying degrees of internal echoes and debris. Sonographically, liver abscesses are often ill defined. The lack of definition can make liver abscesses less conspicuous on sonographic images than on contrast CT scans.

On contrast CT scans, hepatic abscesses are generally hypodense lesions, either unilocular with smooth outer margins or highly complex with internal septations and irregular margins. Pyogenic abscesses tend to be better defined on CT scan than on sonography because contrast enhancement highlights the unenhancing lesion. Rim enhancement and gas-containing abscesses are relatively uncommon.

Early detection by sonography or CT scan, improved antibiotic therapy, guided percutaneous fine needle aspiration for diagnosis, and percutaneous drainage treatment
have greatly improved the clinical management of these patients, reducing the need of surgery treatment and declining the mortality rate.

Antibiotic treatment should be started before or during the drainage procedure. Imaging with sonography or CT immediately after catheter placement may be useful to document catheter location.

Complications of percutaneous hepatic abscess drainage are uncommon but include septicaemia caused by manipulation of the abscess cavity, haemorrhage, pneumothorax, empyema, catheter dislodgment, and free intraperitoneal spill.

*In a patient with a pyogenic hepatic abscess of unknown etiology, the clinician should suspect and look for silent colonic cancer. (Figures 15-16).*

**ACUTE HEPATITIS**

At histologic analyses, hepatitis show cellular alterations with differing penetration of periportal hepatocellular necrosis, Kupffer cell mobilization, and portal inflammation of plasma cells, depending on the underlying infectious, toxic, or autoimmune cause (Figure 17).

These inflammatory entities can be self-limiting, progress to segmental scarring, or end in an overall cirrhotic state.

The acute variant of hepatitis lasts less than 6 months; chronic hepatitis represents any inflammatory condition of the hepatic parenchyma that does not show signs of regression for periods longer than 6 months.

**Viral hepatitis** still remain the most common causes of liver disease worldwide. Six hepatotropic viruses have been identified: picornavirus (hepatitis A), hepadnavirus (hepatitis B), flavivirus (hepatitis C), hepatitis E virus, togavirus (hepatitis F), and GB virus C (hepatitis G).

A vast array of other viruses may also produce hepatitis, including herpes viruses, yellow fever virus, rubella virus, Coxsackie virus, and adenovirus. Although these viruses can be distinguished by their molecular and antigenic properties, all types of viral hepatitis produce clinically similar illnesses. These illnesses range from asymptomatic, unapparent infections to fulminant, fatal acute infections, and from subclinical persistent infections to rapidly progressive chronic liver disease with cirrhosis (common to the blood-borne types [HBV and hepatitis C or D virus]).

Acute forms of viral hepatitis usually show hepatomegaly with an edematous liver capsule and distinct necrotic areas that result in irregularities of the liver contour. In its fulminant
variant, acute hepatitis causes extensive necrosis with shrinkage of the entire organ and significant loss of parenchymal volume. (Figure 19-20)

Chronic forms show fibrosis and possible cirrhosis of the liver. Ascitis and splenomegaly can accompany any state of chronic hepatitis.

The imaging features of acute hepatitis are nonspecific, and the diagnosis is usually based on serologic, virologic, and clinical findings. Probably the most important role of radiology in patients with suspected hepatitis is to help rule out other diseases that produce similar clinical and biochemical abnormalities, such as extrahepatic cholestasis, diffuse metastatic disease, and cirrhosis.

At US, in acute hepatitis, the liver is often enlarged and may demonstrate a diffuse decrease in parenchymal echogenicity, causing a relative increase in the echogenicity of the portal vein walls ("starry night" pattern). A normal liver echotexture does not exclude the diagnosis of acute hepatitis. Gallbladder mural edema is commonly founded (Figure 18).

In chronic hepatitis, the hepatic inflammation and necrosis continue for at least 6 months. Complications that occur during end-stage chronic hepatitis include ascites, edema, variceal bleeding, hepatic encephalopathy, coagulopathy, hypersplenism, and development of hepatocellular carcinoma.

Multidetector CT of acute and fulminant courses of hepatitis shows generalized hepatomegaly combined with peripheral edema. Furthermore, nonenhanced multidetector CT can show heterogeneous attenuation patterns. The overall hepatic parenchymal attenuation is usually equal to or less than that of the spleen. Contrast enhanced multidetector CT can demonstrate irregular perfusion with heterogeneous regions of attenuation. As with multidetector CT, hepatic MR imaging shows general hepatomegaly combined with edema of the liver capsule. In addition, regions of hyperintensity can be seen surrounding the portal venous branches on T2-weighted images. An enhancement pattern similar to that seen at multidetector CT can be seen on dynamic and multiphase contrast-enhanced MR images.

AMOEbic LIVER Abscess

*Entamoeba histolytica* is an important worldwide cause of liver abscess, especially in tropical and subtropical regions. *E. histolytica* are ingested as cysts and pass to the colon where vegetative trophozoites enter the intestinal mucosa, causing colonic ulcers. Amoebae then enter the portal vein and are swept to the liver. In the liver, the amoebae block small portal radicles, release enzymes, and cause focal inflammatory lesions.

Hepatic abscess is the most common extraintestinal complication of amebiasis and occurs in approximately 8.5% of all patients with amoebic infection.
Single or multiple abscesses may then be formed, although in most patients single abscess is found.

The preferred site for abscess formation is superoanteriorly in the right lobe of the liver. The right hemidiaphragm may be elevated and fixed. Pleural effusion is frequent (Figures 21-22).

There is scant correlation between the appearance of the liver abscess and evidence of active colonic infection. Long latent intervals have been documented between intestinal infection and the onset of an abscess.

The gradual onset of fever, malaise, and right upper quadrant abdominal pain is the usual presentation for a patient with an amoebic abscess of the liver. Somewhat surprisingly, patients with amoebic abscess are often more acutely ill than patients with pyogenic abscess. Amoebic liver abscess usually presents as a primary condition, causing the patient to seek medical aid.

Jaundice is unusual and, if present, suggests that the abscess has compressed a major bile duct. Only a few patients have concomitant evidence of amoebic colitis, and cysts are found in the stool in a minority of patients (Figure 27). Leukocytosis and anemia may be present.

Amoebic abscess should be considered in any patient who has resided in or travelled to an endemic area and in whom a hepatic filling defect is found on an imaging study.

The indirect hemagglutination test indicates tissue invasion by amoebae and in almost always indicative, although not diagnostic, of liver involvement.

For treatment, metronidazole is the drug of choice. Because amebicidal therapy is generally highly effective, catheter drainage of amebic abscess is rarely necessary in patients with amoebic abscesses (Figure 28).

Both CT scans and sonography are sensitive techniques for the diagnosis of hepatic amoebic abscesses. The imaging features alone, however, are often non-specific. In many patients, amoebic abscesses cannot be reliably differentiated from pyogenic abscesses or other complex cystic lesions, although epidemiological and clinical information may suggest the diagnosis.

Sonographic reliable features of amoebic abscesses are rounded or oval shape and a hypoechoic appearance with homogeneous, low-level internal echoes.

On contrast CT scans, amoebic abscesses are most often rounded, well-defined lesions with low attenuation values indicating complex fluid. An enhancing wall (3-15 mm) and a peripheral zone of edema around the abscess are common but not universal features. The appearance of the central abscess cavity is quite variable. There may be
multiple septations or fluid-debris levels. Rarely, gas bubbles or areas of haemorrhage are identified within the abscess cavity. The outer margin of an amoebic abscess may be either smooth or irregular. Extrahepatic extension of amoebic abscess (chest wall, pleural, or adjacent visceral involvement) is relatively common and is often demonstrated by CT scans.

(Figures 23-24-25-26)

**Differential diagnosis** includes calcified chronic haematoma, tuberculoma, hydatidosis, hepatic neoplasms, and calcified metastasis of tumors that produce mucus or bone and cartilaginous tissue.

**HEPATIC BRUCELLAR ABSCESS**

Liver abscess is uncommon in patients with active brucellosis. Brucellar hepatic abscess results from the caseous necrosis of granulomatous tissue induced by persistent *Brucella* in macrophages.

In Mediterranean countries, brucellosis is primarily due to *Brucella melitensis*.

Non-specific or granulomatous hepatitis occurs in more than 50% of cases of brucellosis. The hepatic brucelloma (or hepatic abscess by brucellosis or necrotizing pseudotumoral granuloma) is a rare type of hepatic manifestation by *Brucella* and is noted only in the 1.7% of the patients affected by brucellosis. Nevertheless, in these patients, the involvement of the liver by brucellosis is almost constant and is asymptomatic.

The infrequent and rarely specific clinical findings are fever, pain in the right upper quadrant of the abdomen, asthenia, and loss of weight.

The biological tests show in most of the cases a specific inflammatory syndrome and the liver function tests are most of the time within the normal limits.

The Wright's and rose Bengal tests are often positive.

The images obtained by US and CT are characteristic. They show a pseudo-tumoral lesion, more often single, hypoechoic and hypodense, heterogeneous and in its centre a single or multiple calcium deposits. In this lesion, fluid is collected in one or more loculi, whose walls enhance following the intravenous injection of iodinated contrast medium.

The central calcium deposit should bring to mind an abscess by brucellosis and exclude other lesions producing an intrahepatic tumoral or pseudo-tumoral mass (Figures 29-30-31).
HEPATIC HYDATID DISEASE

Hydatid disease (HD) is a unique parasitic disease that is endemic in many parts of the world.

Dogs or other carnivores are definitive hosts. Humans may become intermediate hosts through contact with a definitive host (usually a domesticated dog) or ingestion of contaminated water or vegetables.

An HC has three layers. The outer layer, or pericyst, consists of modified host cells, fibroblasts, giant cells, and eosinophils, which together form a rigid protective layer only a few millimetres thick. The pericyst represents the response of the host to the parasite. The middle laminated membrane is thin, acellular and is about 2 mm thick. Disruption of the laminated membrane predisposes to infection. The inner germinal layer is thin and translucent. Scolices, the infectious embryonic tapeworms, develop from an outpouching of the germinal layer known as the brood capsule.

Hydatid disease primarily affects the liver and typically demonstrates characteristic imaging findings.

The liver is the most frequent site of involvement (75% of cases) and the right lobe is the most frequently involved portion of the liver.

Imaging findings in hepatic hydatid disease depend on the stage of cyst growth.

Calcification is seen at radiography in 20-30% of HCs and usually manifests with a curvilinear or ringlike pattern representing calcification of the pericyst (Figures 32) During the natural evolution toward healing, dense calcification of all components of the cyst occurs.

Dense calcification of the pericyst and cyst contents is common in end-stage hydatid disease and implies the death of the parasite (Figures 33).

Ultrasonography (US) is the most sensitive modality for the detection of cystic membranes, septa, and hydatid sand within the cyst. Multivesicular cysts manifest as well-defined fluid collections in a honeycomb pattern with multiple septa representing the walls of the daughter cysts. Daughter cysts appear as cysts within a cyst. Membranes may appear within the matrix as serpentine linear structures, a finding that is highly specific for hydatid disease (Figure.

When the matrix fills the cyst completely, a mixed echogenic pattern is created that mimics a solid mass.

<p>| Sonographic Caremani Classification |</p>
<table>
<thead>
<tr>
<th>Type 1</th>
<th>Simple</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type 2</td>
<td>Liquid cyst with multiple septa and daughter vesicles</td>
</tr>
<tr>
<td>Type 3</td>
<td>Liquid cyst with floating membrane</td>
</tr>
<tr>
<td>Type 4</td>
<td>Solid cyst with heterogeneous pattern</td>
</tr>
<tr>
<td>Type 5</td>
<td>Completely calcified cyst</td>
</tr>
</tbody>
</table>

Types 1 and 2 always with infecting capability
Types 4 and 5 inactive and with death parasite
Type 3 detachment of the endocyst with suffering of the parasite, may progress to type 2 or type 4

Computed tomography (CT) best demonstrates cyst wall calcification and cyst infection. Cyst fluid usually demonstrates water attenuation (3-30 HU). Daughter vesicles manifest as round structures located peripherally within the mother cyst. (Figures 35-36) (Figures 37-38)

Intrahepatic complications include cyst rupture and infection (Figure 39). Although rupture may be related to minor trauma, the natural history of hepatic hydatid cysts implies rupture as a complication in 50-90% of cases.

Three different types of cyst rupture have been described: contained, communicating, and direct. Contained ruptures occur when the endocyst ruptures but the pericyst remains intact. Endocyst detachment is seen at cross sectional imaging as floating membranes within the cyst.

Communicating rupture implies passage of the cyst contents into the biliary radicles that have been incorporated into the pericyst.

Direct rupture occurs when both the pericyst and endocyst rupture, allowing spillage of hydatid material into the peritoneal cavity (Figure, pleural cavity, hollow viscera, abdominal wall, and so on. (Figure 42)

**Infection** occurs only after rupture of both the pericyst and endocyst (communicating and direct rupture), which allows bacteria to pass easily into the cyst (5-8% of cases). At clinical examination, infection usually manifests as a hepatic abscess. US and CT findings are similar to those in other hepatic abscesses. Findings that suggest infection include a solid appearance, a mixed pattern with fluid and solid elements, internal echogenic foci, and air or air-fluid levels within the cyst.
CT is the modality of choice for demonstrating cyst infection. Contrast-enhanced CT may reveal the typical high-attenuation rim representing abscesses surrounding the lesion. CT also most clearly depicts gas or air-fluid levels within the cyst.

**Exophytic growth.** The two most common routes of exophytic growth are the bare area of the liver and the gastrohepatic ligament.

**Peritoneal seeding.** Peritoneal echinococcosis is almost always secondary to hepatic disease and usually goes undetected until cysts are large enough to produce symptoms.

**Biliary communication.** Has been described in up to 90% of hepatic cysts however, frank rupture into the biliary tree occurs in only 5-15% of cases. Jaundice, fever, and chills are the most frequent symptoms related to biliary obstruction and cholangitis.

Percutaneous drainage of uncomplicated hepatic hydatid cysts combined with albendazole can be performed safely and results in the disappearance of the cyst. The efficacy of percutaneous drainage is similar to that of standard treatment with cystectomy, in terms of reducing the size of the cyst and causing its disappearance over a period of up to two years.

Generally, percutaneous treatment entails drainage and instillation of a sclerosing, scolicidal agent. Pre-treatment with albendazole is preferred. Precautions to prevent allergic and anaphylactic reactions are necessary.

The advantages of percutaneous drainage include a shorter hospital stay and a lower complication rate.

*Surgical treatment* is indicated in large cysts (> 10 cm), peripheral cysts, infected cysts or communicating with the biliary tree.

**MYCOBACTERIUM INFECTION (Mycobacterium tuberculosis)**

Tuberculosis remains very important infectious disease. Increasing incidence is related with migratory changes from high incidence areas, the human immunodeficiency virus (HIV) and the relaxation of the prevention and control programs.

More than 35% of cases tuberculosis is the diagnostic disease of AIDS although his incidence has decreased. The frequency of tuberculosis is especially high in intravenous drug addict and in patients that has to be in prison.

The treatment with highly active antiretroviral therapy (HAART) in patients infected with HIV has improved their immune response diminishing the probability to develop opportunistic infections, especially tuberculosis.
INFECTED HEPATIC METASTASES

Hepatic metastases may be infected when they have a high necrotic/hypoxic component favouring anaerobic environment ideal for bacterial growing, especially in immunosuppressed patients and patients with chemotherapy treatment.

POST-SURGICAL HEPATIC INFECTION

General abdominal surgery and specific hepatic surgery may produce hepatic infection, usually as a single hepatic seroma/haematoma or multiple hepatic abscesses when the surgery is focused in the biliary tract producing as a complication an ascending cholangitis.

Infected hepatic haematoma (Figure 43)

Post-surgical hepatic abscess (Figure 45)

POST-PERCUTANEOUS TREATMENT OF HEPATIC TUMOR

Patients diagnosed with primary hepatic malignancies or metastases to the liver remain a difficult population to treat. A small percentage of these people can undergo surgical resection or transplantation. In the remaining nonsurgical patients, minimally invasive techniques as radiofrequency ablation (RFA), either to cure or palliate these patients are a requirement for complete cancer care.

Although percutaneous radiofrequency ablation is considered a safe treatment technique, a variety of complications have been reported. Of these, liver abscess is one of the most common complications.

Up to 1 week after ablation, the necrotic cavity shows variations of density by CT and a wide variation from high to low signal intensity on T1- and T2-weighted images by MR. The signal intensity on T1-weighted images is determined by the stage of the hemorrhage, whereas the signal intensity on T2-weighted images is influenced by the presence of either coagulative necrosis or liquefactive necrosis.

In the immediate post ablation period, a smooth rim surrounding the necrotic cavity can be observed with moderate to intense enhancement on arterial-dominant phase images in dynamic CT and MR studies. The rim surrounding the necrotic cavity immediately after
Ablation shows intense inflammatory reaction and hemorrhage. By 1 week post procedure the rim of enhancement generally ranges from 1 to 3 mm in thickness. Gradually, the inflammatory reaction and hemorrhage are replaced by granulation tissue and the thickness of this rim tends to regress progressively, and the extent of enhancement on arterial-dominant phase images also diminishes, until complete disappearance generally by 6 months after ablation.

Gas in the tumors accumulated during the course of the 12-minute ablation session was at a maximum at the end of the ablation procedure. The gas typically dissipated within 20 minutes. Almost all of the gas at the tumor ablation site had dissipated if a post procedural contrast-enhanced CT scan is obtained.

The presence of air in the ablated cavity immediately after ablation represents a common and normal feature and tends to disappear within 1 month.

An ablation zone with gas after 1 month frequently with a peripheral enhancing rim on CT is considered diagnostic of liver abscess in an adequate clinical setting (bacteria were cultured from the aspirate or blood samples, aspirate from the ablation zone had a purulent appearance at macroscopic or microscopic examination; or if associated fever higher than 38.5°C, lasted more than 5 days and leukocytosis without other causes) (Figure 44).

There are multiple preablation conditions as potential risk factors for liver abscess formation after radiofrequency ablation. These included a history of transcatheter arterial chemoembolization, biliary abnormalities, diabetes mellitus, multiple tumors, Child-Pugh class B cirrhosis, tumor with retention of iodized oil, local tumor progression after prior radiofrequency ablation, and relatively large tumors.

Images for this section:
Fig. 1: Patient 62-year-old admitted with epigastric abdominal pain, nausea, dyspepsia, jaundice and cholestasis. Ultrasonographic images shows a large heterogeneous hypoechoic hepatic lesion (9x8x8 cm). Mild intrahepatic biliary dilatation and gallstone disease (not showed).

Fig. 2: Initial contrast enhanced MDCT demonstrates the hypodense heterogeneous lesion with multiple septa and hypo perfusion of adjacent parenchyma. Moderate
dilatation of intrahepatic biliary ducts and occupation of common bile duct by stones and hyperdense material with mural thickening was observed indicating cholangitic changes in biliary obstruction. An endoscopic retrograde cholangiopancreatography (ERCP) was attempted not visualizing the papilla.

![CT images](image)

**Fig. 3:** Follow-up CT after antibiotic treatment in critical care unit shows decrease of the size lesion and parenchymal inflammatory changes, and pneumobilia. Finally ERCP was performed successfully with biliary decompression.
Fig. 4: Postsurgical choledocal stenosis. Patient 76-year-old with history of cholecystectomy and secondary choledocal stenosis. Five months later was admitted with a clinical picture of obstructive jaundice. CT demonstrates dilatation of the intrahepatic biliary tract and the choledochus with abrupt change of calibre compatible with postsurgical stenosis. A laterolateral hepato-duodenostomy was performed.
Fig. 5: Ascending cholangitis with multiple hepatic abscesses associated. The patient was readmitted three years later with a picture of right upper pain, fever, leukocytosis, chills and jaundice. CT shows the presence of multiple hepatic abscesses secondary to an ascending cholangitis with a confluent area localized in segment 7.
Fig. 6: Microabscesses caused by ascending cholangitis by Cryptosporidium. Patient with diagnostic clinical criteria of AIDS presenting upper quadrant abdominal pain and jaundice. CT shows multiple hypodense small foci distributed in the hepatic parenchyma corresponding to hepatic microabscesses secondary to ascending cholangitis confirmed caused by Cryptosporidium parvum.
Fig. 7: Patient with chronic pancreatitis related with alcohol intake and multiple episode of acute pancreatitis. CT scan shows a large lobulated hepatic abscess with multiple septa, occupying caudate lobe and spreading peripherally. It has central hypodensity with ill-defined peripheral enhancement of the contrast material. A percutaneous drainage was performed.
**Fig. 8:** Vesicular empyema complicated with contiguous hepatic abscess. MDCT scan images axial (A) and coronal (B) shows cholelithiasis, inflammatory vesicular changes (mural thickening and involvement of perivesicular fat) with superior and medial hepatic collection with enhancing rim corresponding to an adjacent abscess.
Fig. 9: Hepatic abscess by contiguity of xanthogranulomatous pyelonephritis. The CT scan shows a hypodense lesion with peripheral enhancing rim affecting by contiguity the 6 segment of right hepatic lobe and characteristic features of a xanthogranulomatous pyelonephritis affecting the right kidney.

Fig. 10: Hepatic abscess as a complication of Crohn disease. Patient 16-year-old with Crohn disease with terminal ileitis presenting right flank pain, fiver and peritonitis. A) CT demonstrates the presence of a hepatic collection with peripheral enhancing rim characteristic of a hepatic abscess located in segment 5 of the RHL. Subsequently a sonographic guided percutaneous drainage was performed. B) Pelvic axial images shows inflammatory thickening of the distal ileum.
**Fig. 11**: Cryptogenic hepatic abscess. Patient 73-year-old with fever days ago. A) The sonographic study shows the presence of a large heterogeneous collection (12.5 x 9 x 8 cm) compatible with an abscess occupying posteriors segments of right hepatic lobe and associated pleural effusion. B) CT was performed identifying a large ovoid collection, occupying 7 and 6 segments of RHL, homogeneously hypodense, without peripheral enhancing rim, visualizing tree small hypodense nodules in the same lobe, pleural effusion and widespread of the collection to the posterior abdominal wall.
Fig. 12: Cryptogenic hepatic abscess. A percutaneous drainage guided by sonography was performed. Sonographic image shows the catheter inside the lesion (initially were obtained 350 cc of purulent liquid), with significant diminishing of the size of the abscess. The pus culture was positive for Streptococcus viridans and blood culture were negatives.

Fig. 13: Cryptogenic hepatic abscess. Patient 54-year-old with abdominal pain, fever and diarrhea ten days ago diagnosed as acute gastroenteritis treated with antibiotics unsuccessfully. Endoscopy demonstrates rectal ulcerations. Sonography study shows
the presence of three heterogeneous collections compatible with abscesses occupying superiors segments of right hepatic lobe and associated pleural effusion.

**Fig. 14:** MDCT was performed identifying three large round collection, affecting 8 and 7 segments of RHL, homogeneously hypodense, with peripheral enhancing rim and associated pleural effusion. A percutaneous drainage of the collections (guided by ultrasonography) was performed.

**Fig. 15:** Pyogenic abscess in patient with rectal ulcerated adenocarcinoma initially unknown. Patient 53-year-old admitted with fever, asthenia, weight loss and persistent
diarrheic syndrome. Sonography shows an large hypoechoic lobulated lesion suggesting hepatic abscess and drained percutaneously. Endoscopic study showed a rectal ulcerated adenocarcinoma.

**Fig. 16:** Pyogenic abscess in patient with rectal ulcerated adenocarcinoma initially unknown. A) CT scan obtained after percutaneous drainage demonstrates catheter drainage placement and shows the hepatic abscess affecting the majority of RHL. B) Follow-up CT scan shows diminished size of the abscess.

**Fig. 17:** Acute alcoholic hepatitis with picture of Severe Hepatic insufficiency. Patient 43-year-old admitted at hospital with alcohol intake and cutaneous mucous jaundice and vomiting. CT after intravenous contrast Administration in portalvenous phase
shows marked hepatomegaly with patched hepatic perfusion identifying low attenuation extensive areas and lesser higher attenuation areas.

Fig. 18: Toxic acute hepatitis. Patient with clinical diagnostic criteria of AIDS affected of tuberculous infection that developed hepatic acute toxicity caused by Isoniazid. Ultrasonography shows a diffuse and severe thickening of the gallbladder wall.
Fig. 19: Fulminant acute viral hepatitis. Patient 31-year-old with hemodynamic instability and metabolic acidosis. Axial images (A) and coronal reconstructions (B) of contrast enhanced MDCT in portal venous phase shows severe global hepatomegaly, periportal edema, gallbladder edema, and multiples hypodense focal hepatic lesions. Pathologic diagnosis was acute hepatitis caused by Epstein Bar virus combined with lymphoid infiltrates, producing fulminant hepatic failure and the death of the patient.
**Fig. 20:** Fulminant acute viral hepatitis. Patient 31-year-old with hemodynamic instability and metabolic acidosis. Axial images (A) and coronal reconstructions (B) of contrast enhanced MDCT in portal venous phase shows severe global hepatomegaly, periportal edema, gallbladder edema, and multiples hypodense focal hepatic lesions. Pathologic diagnosis was acute hepatitis caused by Epstein Bar virus combined with lymphoid infiltrates, producing fulminant hepatic failure and the death of the patient.
Fig. 21: Patient 28-year-old traveller from India one year ago submitted with fever, chills, vomiting, diarrhea and right upper quadrant pain. Axial and coronal MDCT images demonstrates a large lesion (10 cm) located anterior and upper in the RHL (VIII segment) presenting a peripheral thick rim. Antibiotic therapy and percutaneous drainage guided by sonography (B) were performed obtaining purulent content. Final diagnosis by serological test was abscess caused by Entamoeba histolytica. Mild bilateral pleural effusion and ascitis were detected.
**Fig. 22:** Patient 28-year-old traveller from India one year ago submitted with fever, chills, vomiting, diarrhea and right upper quadrant pain. Axial and coronal MDCT images demonstrate a large lesion (10 cm) located anterior and upper in the RHL (VIII segment) presenting a peripheral thick rim. Antibiotic therapy and percutaneous drainage guided by sonography (D) were performed obtaining purulent content. Final diagnosis by serological test was abscess caused by Entamoeba histolytica. Mild bilateral pleural effusion and ascitis were detected.
Fig. 23: Amoebic liver abscess. Patient recent traveller from Filipinas, 28-year-old with fever, chills and right upper quadrant pain treated with antibiotherapy. A) Ultrasonography shows an hyperechoic heterogeneous lesion (6 x 4,8 cm), with rounded shape, surrounded by hypoechoic halo and localized in five segment of right hepatic lobe. Initially was suspected solid tumour and CT scan was performed. B) CT shows an hypodense lesion with peripheral enhancement in arterial phase. There are also perfusion peripheral disorders being compatible with an hepatic abscess (7 x 6 x 6 cm), affecting five and eight segments of right hepatic lobe. Inflammatory changes of hepatic parenchyma are associated with the lesion.
Fig. 24: Amoebic liver abscess. A) Nonenhanced CT shows characteristic hyperdense areas correlated with the hyperechoic centre visualized by ultrasound (because of hemorrhage into the cavity) and peripheral hypodense ring. B) Contrast enhanced CT in later phase continues demonstrating peripheral enhancing ring of the lesion and central hyperdense focus.

Fig. 25: Ultrasonography appearance one week later. The lesion has become homogeneously hypoechoic, surrounded by echogenic peripheral ring. Fine needle aspiration was performed for microbiology study and a percutaneous drainage was placed. It was interpreted as a pyogenic abscess or amoebic abscess beginning combined antibiotic therapy obtaining clinical response in 24 hours disappearing the fever. The drainage was removed because of obstruction in 5 days continuing with medical treatment.
**Fig. 26**: Ultrasonography appearance one week later. The lesion has became homogeneously hypoechoic, surrounded by echogenic peripheral ring. Fine needle aspiration was performed for microbiology study and a percutaneous drainage was placed. It was interpreted as a pyogenic abscess or amoebic abscess beginning combined antibiotic therapy obtaining clinical response in 24 hours disappearing the fever. The drainage was removed because of obstruction in 5 days continuing with medical treatment.
**Fig. 27:** Hepatic abscess by *Entamoeba histolytica* and enteritis. Patient 57-year-old with history of hepatic abscess eight years ago, submitted with abdominal hypogastric pain and vomiting. CT images shows hepatic hypodense lesion with peripheral enhancing rim (47 mm) located in the VIII segment of the RHL. Associated mural thickening of small bowel loops was detected, consistent with enteritis.

![CT images of hepatic abscess](image)

**Fig. 28:** Patient 44-year-old submitted with epigastric abdominal pain. Ultrasonography detected well-circumscribed hypoechoic lesion with peripheral rim located in the V segment of the right hepatic lobe and cholelitiasis. A fine needle aspiration was performed. Serological final diagnosis was abscess caused by *Entamoeba histolytica* treated with Metronidazol and Paramomicin.

![Ultrasonography images](image)
Fig. 29: Brucellar hepatic abscess or "Hepatic brucelloma". Patient 26-year-old admitted with a two months history of daily fever and right upper quadrant pain. Sonography demonstrates an oval and hypoechoic lesion with a gross central calcification. He occasionally consumed fresh goat cheese.
**Fig. 30:** Brucellar hepatic abscess or "Hepatic brucelloma". A) Nonenhanced CT shows an hypodense lesion with ill-defined borders and central calcification located in right hepatic lobe. The lesion has a slightly central enhancement in arterial phase (B) and in portalvenous phase (C) with a peripheral enhancement in equilibrium phase (D).

**Fig. 31:** Follow-up with ultrasound and CT of the brucellar hepatic abscess or "Hepatic brucelloma" one month after treatment with doxycycline and rifampicin shows diminished hypoechoic lesion by sonography and hypodense (CT in portalvenous phase) with later enhancement (CT equilibrium phase), persisting the coarse central calcification.
**Fig. 32:** Plain films centered of RUQ showing dense calcifications common in end-stage of hydatid disease.

**Fig. 33:** Sonographic appearance of multiple hydatid cyst type 4 (solid cyst with heterogeneous pattern) and 5 (completely calcified) of Caremani classification indicating death parasite.
**Fig. 34:** Characteristic appearance of the membranes in a hydatid cyst as a serpentine linear structures, finding highly specific for hydatid disease.
**Fig. 35:** Sonography of a large polilobulated hydatid cyst indicating extracapsular extension.
Fig. 36: CT images of the same patient showing extracapsular partial extension.
Fig. 37: Axial sonography images demonstrating multiple characteristic hypoechoic peripheral daughter cysts.

Fig. 38: Axial and coronal CT images of the same patient demonstrating partial thin peripheral calcification and daughter cysts.
Fig. 39: Axial CT images demonstrating multiple daughter cysts two of them with extracystic location as an indication for surgery.
**Fig. 40:** Axial MDCT images showing a very large hydatid cyst affecting right hepatic lobe demonstrating multiple septa.
Fig. 41: Coronal MPR of the same patient showing a very large hydatid cyst affecting right hepatic lobe demonstrating multiple septa.
**Fig. 42:** Multiple peritoneal invasion of right hepatic hydatid cyst.

**Fig. 43:** Infected hepatic haematoma secondary to surgical resection of a metastases. Ultrasonography image showing an heterogeneous hypoechoic lesion.
Fig. 44: Infected HCH treated with radiofrequency ablation detecting bubbles of gas in clinical setting of fever six days ago, right upper quadrant pain and leukocitosis.
Fig. 45: Pyogenic hepatic abscess secondary to surgical resection of a metastases in VII segment of RHL. CT in venous phase demonstrate gas formation.
Fig. 46: Disseminated tuberculosis in AIDS patient. Patient with clinical diagnostic criteria of AIDS submitted with fever and abdominal pain. Axial MDCT images demonstrate splenomegaly and hepatomegaly detecting multiple small hypodense foci affecting the spleen, the liver parenchyma and also the kidneys.
Conclusion

Conclusions

• Septic cholangitis secondary to biliary stones, neoplasm or stenosis of the biliary tract may produce multiple hepatic abscesses.
• In a patient with anaerobic liver abscesses, the clinician should suspect and look for silent colonic cancer. A positive response to medical management does not rule out this possibility.
• Hydatid cyst may be infected producing a clinical picture and radiological findings totally equivalent of a hepatic abscess.
• Puncture, aspiration, injection and reaspiration (PAIR) are a safe and efficacy technique for percutaneous treatment of hepatic hydatid cyst.
• The diagnosis of brucellar hepatic abscess may be made on the basis of the characteristic sonographic features and positive Brucella agglutination tests.

It's important recognizes the imaging features and clinical setting (signs, symptoms, biochemical abnormalities, epidemiological data and immunologic condition) of the inflammatory-infectious liver disease for an accurate early diagnosis enabling an appropriate management and treatment.

Personal Information

References


