# Diagnostics of acute pain in abdominal right upper quadrant

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### Abstract

Acute abdomenal pain is a frequent clinical problem encountered in emergency departments that can present a considerable challenge to a junior doctor. We discuss here some common causes of abdominal pain localized in the upper right quadrant, reviewing its clinical and imaging features.

### Key words

acute abdominal pain, ultrasonography, computer tomography

### INTRODUCTION

The term acute abdomen denotes a disorder of sudden onset with a duration of less than 24 hours that is manifested as abdominal pain and associated with gastrointestinal symptoms. Patients with acute abdominal pain are usually first seen at an accident and emergency department and present a considerable challenge to the junior doctor.

Acute abdomen with pain localized in an abdominal quadrant can be classified as pain in the right upper, left upper, right lower, and left lower abdominal quadrant. The most common causes of acute pain in the abdominal quadrants are listed in Table 1.

Right upper quadrant	Acute calculous/acalculous cholecystitis Liver abscess Spontaneous rupture of hepatic neoplasm Myocardial infarction
Left upper quadrant	Splenic infarction Splenic abscess Gastritis Gastric ulcer
Right lower quadrant	Acute appendicitis Acute terminal ileitis (Crohn's disease) Acute typhlitis Pelvic inflammatory disease Complications of ovarian cyst (hemorrhage, torsion and leak) Endometriosis Ectopic pregnancy
Left lower quadrant	Diverticulitis Epiploic appendagitis

In the right upper quadrant, acute cholecystitis is by far the most common disease. Other important diseases are: liver abscess, spontaneous rupture of a hepatic neoplasm (usually hepatocellular carcinoma), and myocardial infarction. Ultrasonography is the preferred imaging method for evaluating patients with acute right upper abdominal pain [2].

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Received: 2 June 2011; accepted: 18 October 2011

The patient with acute abdominal pain is best dealt with as an emergency, possibly surgical in nature.

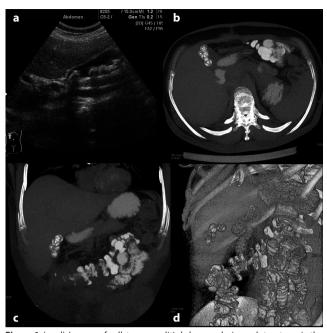
At the forefront of liver diagnostics are the tried and tested methods of anamnesis, inspection, palpation and percussion, or even auscultation. It must be ascertained whether the disease has occurred suddenly, developed gradually, or simply not been noticed up to now. Determining the liver size is considered to be the 'simplest' liver function test and, chronologically speaking, it is also the 'first'. Pain on palpation mainly points to capsular tension as a result of an enlarged liver (acute viral hepatitis, fatty liver, congested liver). In general, the metastatic liver is likewise painful. Liver abscesses may cause (frequently severe) pain on tapping.

Acute cholecystitis. Acute cholecystitis commonly presents with right upper quadrant pain 1-2 hours after eating, and is often accompanied by nausea and vomiting. The pain reaches maximum intensity rapidly and may persist for a prolonged period, often with shoulder radiation.

Clinical features of acute cholecystitis include anorexia, nausea, vomiting, fever with temperatures of 38°-39° C, severe abdominal pain that is initially localized to the epigastrium, and right upper quadrant tenderness. The pain typically lasts longer than 6 hours. Up to 15% of patients notice dark urine and scleral icterus. Most patients with jaundice have stones in the common bile duct at the time of surgery.

Physical examination reveals right upper quadrant subcostal tenderness and pain on inspiration, often with inspiratory arrest (the Murphy's sign). The pain may spread to other areas of the abdomen. Tenderness, guarding, and rebound pain in the area of an inflamed gallbladder are important findings. Laboratory tests frequently reveal leukocytosis (leukocytes usually number 12,000-15,000/ $\mu$ l<sup>3</sup>). Serum bilirubin and alkaline phosphatase levels are elevated in 30%-50% of patients.

Ultrasonography is the diagnostic procedure of choice for a patient with suspected gallstones and acute cholecystitis. A meta-analysis revealed that ultrasonography has a sensitivity of 88%-90% and a specificity of 97%-98% for the diagnosis of gallstones bigger than 2 mm in size [3]. Gallstones are a typical appearance on ultrasound images. They present as hyperechoic, oval structures of high intensity, with an acoustic shadow behind them at the background of a hypoechoic gallbladder.



**Figure 1.** (a – d). Images of gallstones: mulitiple hyperechoic, oval structures in the gallbladder with clear acoustic shadows on a ultrasound image (a); hyperdense, oval stones well visualized on multiple planar reconstructions images in CT (b, c); and 3-D reconstructed CT image enabling precise spatial localization of gallstones (d).

Ultrasound is the examination of choice in evaluating the gallbladder. It can be useful in confirming that the area of maximum tenderness overlies the gallbladder (sonographic Murphy's sign). Even in the absence of gallstones, this finding, in association with thickening of the gallbladder wall (measuring >3 mm) suggests a diagnosis of acalculous cholecystitis; however, a thickened wall in isolation may be due to many other causes, including hypoalbuminaemia and ascites [4].

In addition to detecting gallstones, ultrasonography can be used to identify other causes of right upper quadrant pain, such as hepatic abscess or malignancy, and it may reveal biliary duct obstruction.

In difficult cases, CT may be used to search for gallstones complications. Gallstones are then visible as hyperdense, oval structures of high Hounsfield units located inside the gallbladder. Three-dimentional reconstructed images may be useful to visualize their spatial location.

**Choledocholithiasis.** In the United States, gallstones occur in approximately 10% of persons older than 40 years of age, but the prevalence is significantly higher in women, increasing to 20%-25% in women above the age of older than 50. Fortunately, only 20%-30% of gallstones are symptomatic, with biliary colic being the most common symptom.

Choledocholithiasis may be complicated by cholangitis, characterized by the triad of fever with shaking chills, jaundice, and right upper quadrant colicky pain. Pain may be severe, have a rapid onset, and last 15-60 minutes. It is usually accompanied by nausea and vomiting.

Patients who have stones in the gallbladder or the biliary tree display syndromes that range from acute disease to chronic symptomatic or silent disease. Most gallbladder stones remain silent throughout a person's lifetime.

Biliary pain, whether arising from the gallbladder or the bile duct, is felt initially as a dull sensation in the midline (epigastrium); with the development of inflammation or if the pain continues, it is often referred to the right upper quadrant and occasionally to the right infrascapular region. Acute obstruction of the biliary tree is almost always painful, although not necessarily colicky in the true sense.

Acute inflammation of the gallbladder presents with severe RUQ pain localized to the gallbladder area. The pain can be elicited by pressing the gallbladder with the ultrasound transducer – a positive ultrasound Murphy's sign.

On ultrasound, the gallbladder wall is thickened by more than 2 mm. This is not in itself a specific sign, but characteristically the thickening in acute cholecystitis is symmetrical, affecting the entire wall, and there is an echopoor 'halo' around the gallbladder as a result of oedematous changes. This is not invariable, however, and focal thickening may be present, or the wall may be uniformly hyperechoic in some cases.

Acute viral hepatitis. Most cases of acute hepatitis are caused by one of the hepatotrophic viruses, but drug-induced hepatitis and hepatitis that is secondary to other viruses may at times mimic typical acute viral hepatitis. Classic acute viral hepatitis is caused by one of 5 etiologic agents: hepatitis A virus (HAV), hepatitis B virus (HBV), hepatitis C virus (HCV), hepatitis D virus (HDV), or hepatitis E virus (HEV).

In the course of acute viral hepatitis, the liver becomes enlarged and can be painful. This is where imaging can be of help in determining the extent of liver enlargement. It can be well assessed by ultrasonography or other imaging methods. However, in a clinical suspicion of hepatitis, imaging methods are not of the first use, as they do not provide any other specific information characteristic for the disease.

Hepatocellular carcinoma. Tumors in the liver are largely asymptomatic. Hepatocellular carcinoma (HCC) can present with pain, but other symptoms are nonspecific and include anorexia, weight loss, worsening ascites, and encephalopathy. Screening is recommended for patients with known chronic hepatitis or end-stage liver disease, generally with serum alpha fetoprotein AFP measurements and ultrasound or magnetic resonance imaging (MRI), depending on the patient's risk. The majority of HCC cases develop in the cirrhotic liver. Cirrhosis is the strongest predisposing factor for HCC, with chronic viral hepatitis the commonest underlying etiological cause worldwide [5].

Early detection of HCC is therefore critical to achieve effective treatment and prolong survival [6]. HCC appearances on B-mode US are variable. Small lesions are usually hypoechoic, but larger lesions may demonstrate heterogeneous echotexture due to necrosis and fibrosis.

HCC is now one of the commonest causes of cancer death worldwide and is the fifth most common cancer worldwide [7]. Cholangiocarcinoma accounts for 3% of all gastrointestinal cancers but has increased relatively rapidly worldwide[8].

Currently, B-mode US is recommended in the screening of patients at risk of HCC, including patients with hepatitis B and cirrhosis [9]. In experienced hands, B-mode US can detect 80%-95% of lesions 3-5 cm in diameter and has 60%-80% sensitivity in the detection of lesions of 1 cm [10]. Although the identification of a mass with B-mode US varies from 37% to 87% [11], B-mode US is a highly sensitive method for confirming biliary duct dilatation, localization of the site of obstruction and excluding gallstones [12]. Alphafetoprotein, the most commonly used serum tumor marker, is unfortunately not a dependable biomarker for diagnostic and prognostic purposes, having poor sensitivity, specificity, and positive predictive value, emphasizing the importance of high quality imaging techniques [13]. A recent prospective study comparing imaging findings with pathological examination of the explanted liver in a pretransplant population, found that US, MRI and CT had similar sensitivities for HCC detection on a lesion-by-lesion basis, although US performed slightly better on a patient-bypatient basis [14].

Detection of small hepatocellular carcinoma in advanced stage of liver cirrhosis with a shrunken liver is very difficult because the ultrasonographic window is usually limited and the hepatic echotexture is heterogeneous due to the presence of fibrosis, fatty infiltration, parenchymal necrosis, and the myriad of cirrhotic regenerative nodules [15].

Liver metastases. Metastatic tumors are the commonest malignant lesions affecting the liver. Metastasis is a secondary focus of disease caused by haematogenic, lymphogenic or ductal transport of living or dead matter from the primary focus of disease. Liver metastasis develops via the portal vein (particularly in carcinomas of the gastrointestinal tract), via the hepatic artery (e. g. from the lung, breast, esophagus, pancreas, and in melanomas) or via retrograde lymphatic permeation and extension along the vascular lumen. This occurs in 30-35% of all malignancies and in 45-50% of abdominal tumors. In 95-97% of cases, liver metastases cause malignant hepatic lesions.

The occurrence and growth of liver metastases are accompanied by increasing lassitude and malaise, a feeling of weakness, febrile attacks, upper abdominal pain, in appetence, night sweats and weight loss. The spleen may also be enlarged. Jaundice usually develops as the tumor continues to grow.

Metastases can be detected by ultrasonography from a size of >0.5 cm. Due to their high water content, they appear as hypoechoic lesions. With continued growth, central echo amplifications occur due to regressive tissue changes. In metastasis, a halo has a specificity of 86% and a sensitivity of 88% a finding which, however, also applies to benign tumors. Most metastases in CT are hypodense prior and subsequent to the application of contrast medium (Fig. 2).

Sensitivity is 92% in foci of >15 mm, but only 55% in smaller ones. When there is concomitant fatty degeneration, CT may be inferior to sonography for the detection of metastases. The best results were achieved by using spiral CT with a sensitivity of 92-93%; all metastases larger than 6 mm could be identified.

**Liver abscess.** The term liver abscess describes a circumscribed, often encapsulated, purulent inflammation with necrosis of the local parenchyma caused by a multitude of pathogens (bacteria, protozoa, helminthes) and fungi.

Patients with liver abscess usually present with fever, chills, anorexia, fatigue, or other symptoms such as nausea, vomiting, right-upper-quadrant pain, diffuse abdominal pain, pleuritic chest pain, and jaundice [16]. Fever, the most common symptom, reportedly occurs in 70–95% of cases. The most common laboratory finding in these patients is leukocytosis. Alkaline phosphatase levels 2–3 times the upper limit of normal are also common [17].



Figure 2. CT portal phase axial image showing multiple, diffuse, hypodense mass lesions in the liver with clear rim-enhancement consistent here with multiple metastatic tumors from gastrointestinal tract.

Elevated transaminase levels and hypoalbuminemia are also commonly observed.

A US image of liver abscess usually shows a low echoic to mixed-echoic lesion. The margin may be blurred or irregular due to inflammation of surrounding areas. During the early stage, mixechoic parenchyma mass lesions are common, but significant tissue liquefaction is rare [18, 19].

CT scans reveal low attenuation in all liver abscesses, and 91.7% of the enhanced lesions had a rim-shaped enhancement in the abscess wall.

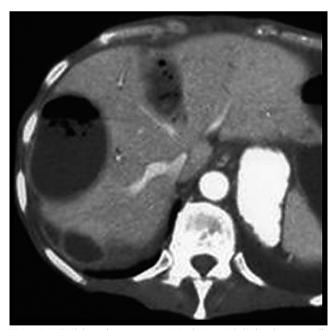


Figure 3. Multiple hypodense, gas-containing lesions with thick, enhancing walls seen on a portal phase CT axial image after contrast administration – typical features of liver abscesses.

Honeycomb-like, grid-like or strip-like enhancements reportedly occur in 75% of cases. The CT findings of hepatic abscess are usually unremarkable, and diagnosing abscess is rarely difficult [20].

### Journal of Pre-Clinical and Clinical Research, 2011, Vol 5, No 2

## REFERENCES

- 1. Marincek B. Nontraumatic abdominal emergencies: acute abdominal pain: diagnostic strategies. Eur Radiol 2002;12:2136–2150.
- Cooperberg PL, Gibney RG. Imaging of the gallbladder.1987. Radiology 1987;163:605–613
- Shea JA, Berlin JA, Escarce JJ, Clarke JR, Kinosian BP, Cabana MD, Tsai WW, Horangic N, Malet PF, Schwartz JS, et al. Revised estimates of diagnostic test sensitivity and specificity in suspected biliary tract disease. Arch Intern Med 1994;154:2573-2581.
- 4. Patriquin HB, Di Pietro M, Barber FE, Teele RL. Sonography of thickened gallbladder wall: causes in children. Am J Roentgenol 1983;141:57-60.
- Colombo M. Risk groups and preventive strategies. In: Berr F, Bruix J, Hauss J, Wands J, Wittekind C, edit. Malignant liver tumors: basic concepts and clinical management. Falk symposium: Kluwer Academic Publishers 2003;67-74.
- 6. Taura N, Hamasaki K, Nakao K, Ichikawa T, Nishimura D, Goto T, Fukuta M, Kawashimo H, Motoyoshi Y, Shibata H, Eguchi K. Clinical benefits of hepatocellular carcinoma surveillance: a single-center, hospital-based study. Oncol Rep 2005;14:999-1003.
- 7. Bosch FX, Ribes J, Borras J. Epidemiology of primary liver cancer. Semin Liver Dis 1999;19:271-285.
- Khan SA, Toledano MB, Taylor-Robinson SD. Epidemiology, risk factors, and pathogenesis of cholangiocarcinoma. HPB (Oxford) 2008;10:77-82.
- 9. Ryder SD. Guidelines for the diagnosis and treatment of hepatocellular carcinoma (HCC) in adults. Gut 2003;52 Suppl 3:iii1-iii8.
- Colombo M, de Franchis R, Del Ninno E, Sangiovanni A, De Fazio C, Tommasini M, Donato MF, Piva A, Di Carlo V, Dioguardi N. Hepatocellular carcinoma in Italian patients with cirrhosis. N Engl J Med 1991;325:675-680.

- Neumaier CE, Bertolotto M, Perrone R, Martinoli C, Loria F, Silvestri E. Staging of hilar cholangiocarcinoma with ultrasound. J Clin Ultrasound 1995;23:173-178.
- 12. Saini S. Imaging of the hepatobiliary tract. N Engl J Med 1997;336:1889-1894.
- Gomaa AI, Khan SA, Toledano MB, Waked I, Taylor-Robinson SD. Hepatocellular carcinoma: epidemiology, risk factors and pathogenesis. World J Gastroenterol 2008;14:4300-4308.
- 14. Glockner JF. Hepatobiliary MRI. current concepts and controversies. J Magn Reson Imaging 2007;25:681-695.
- Kanno T, Kurioka N, Kim S, Tamori A, Kim K, Oka H, Kuroki T, Mizoguchi Y, Kobayashi K. Implications of hyperechoic lesions in small hepatocellular carcinoma. Gastroenterol Jpn 1989;24:528—534.
- Lederman ER, Crum NF. Pyogenic liver abscess with a focus on Klebsiella pneumoniae as a primary pathogen: an emerging disease with unique clinical characteristics. Am J Gastroenterol 2005;100:322– 331.
- Wong WM, Wong BC, Hui CK, Ng M, Lai KC, Tso WK, Lam SK, Lai CL. Pyogenic liver abscess: retrospective analysis of 80 cases over a 10-year period. J Gastroenterol Hepatol 2002;17:1001–1007.
- Mortele KJ, Segatto E, Ros PR. The infected liver: radiologic-pathologic correlation. Radiographics 2004;24:937–55.
- Ralls PW, Barnes PF, Radin DR, Colletti P, Halls J. Sonographic features of amebic and pyogenic liver abscesses: a blinded comparison. Am J Roentgenol 1987;149:499–501.
- Wang CL, Guo XJ, Qiu SB, Lei Y, Yuan ZD, Dong HB, Liu HA. Diagnosis of bacterial hepatic abscess by CT. Hepatobiliary Pancreat Dis Int 2007;6:271–275.