

Case Report
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The Clinical Spectrum of Liver Masses: Know Before You Judge

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Received: October 18, 2024; **Accepted:** October 24, 2024; **Published:** November 01, 2024

Introduction

Liver Masses present a common clinical dilemma, particularly with the wide spread use of cross-sectional imaging in the Emergency Departments. The accurate and reliable determination of the nature of the liver mass is critical, not only to reassure individuals with benign lesions but also to ensure that malignant lesions are diagnosed correctly. In general, Liver Mass can be divided into two main categories: benign lesions and malignant lesions. Benign masses are usually inflammatory masses including an abscess which can be divided into amebic and pyogenic and truly tumor masses which can be divided into malignant or benign tumors.

Brief Case Presentation

A 54-year-old with compensated alcohol-related cirrhosis and chronic Hepatitis C infection presented with a palpable liver mass, CT and MRI showed a mass that was concerning for Hepatocellular carcinoma (HCC) with diffuse infiltration into the right hepatic lobe and complete thrombosis of the entire portal system (Figure 1, 2, 3).

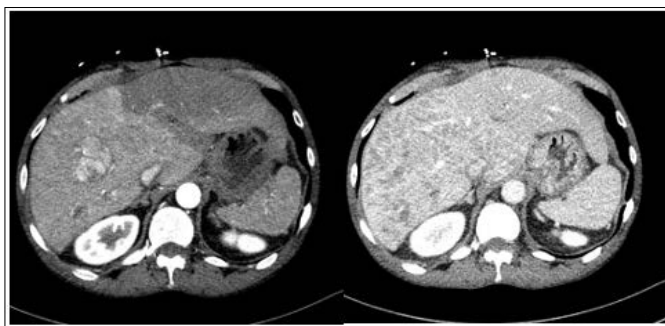


Figure 1: CT with Arterial and Venous Phase Demonstrates an Arterially Enhancing Mass in Segment 7/8 with Washout

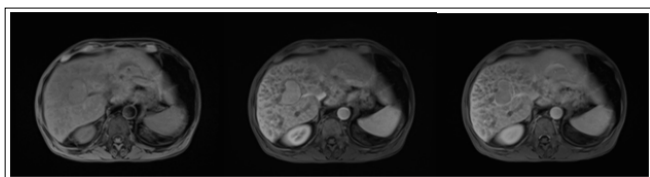


Figure 2: MRI with T1 Pre-Contrast, Arterial Phase, and Venous Phase Demonstrate an Arterially Enhancing Mass in Segment 7/8 with Washout and Enhancing Pseudo Capsule, LI-RADS 5.

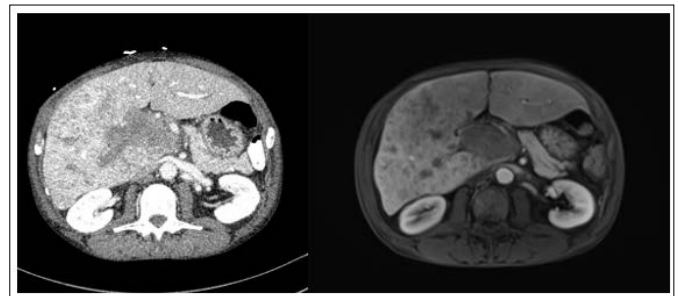


Figure 3: CT and MRI with Portal Venous Phase from the Same Patient Reveal an Expanded Portal Vein with Enhancing Intraluminal Material, Compatible with Tumor Thrombus

Discussion

Patients with liver lesions that are concerning for an abscess most likely have an underlying infectious process and should be categorized into amebic or pyogenic in etiology (Diagram 1). Hepatic abscesses (Figure 4) can occur from different routes including hematogenous spread of infection via the portal vein or hepatic arteries, biliary spread of infection from ascending cholangitis or cholecystitis, or direct inoculation in the setting of penetrating trauma or iatrogenic following a procedure. The potential complications of such a process could include hepatic vein thrombosis (incidence of 22%), portal vein thrombosis (incidence of 24%), sepsis, or rupture into the right subphrenic space, abdominal cavity, pericardium, or gastrointestinal tract.

Symptoms accompanying hepatic abscesses can be vague. Common symptoms include fever, Pain in the right upper abdominal quadrant, fatigue, weakness, weight-loss, and diarrhea. Physical exam findings include jaundice (10-20%), dullness to percussion over the right lower lobe, leukocytosis (90%), normocytic, normochromic anemia (70%), elevated alkaline phosphatase, mildly elevated hyperbilirubinemia (mild, as well as diarrhea which can occurring in 30% of abscess from metastatic infections.

The diagnosis of abscesses is usually made on cross-sectional imaging and confirmed with aspiration. Diagnosis of amebic liver lesions includes serology (100%), stool (cyst of Entamoeba histolytic (25%), or trial of chloroquine. Diagnosis of pyogenic liver lesions including positive aspiration or positive blood cultures (25%). Most bacterial agents causing abscesses in the setting

are polymicrobial include gram-negative aerobic and anaerobic organisms such as *Escherichia coli*, and *Klebsiella pneumoniae* as well as gram positive infections including streptococci or enterococci infections. Hypervirulent strains can cause more severe infections particularly Asian strains that have a predilection for *Bacteroides* infection. Parasitic infections in patients from developing countries including amoebic hepatic abscess that have a non-odorous, anchovy paste appearance of drained contents (Figure 5) and echinococcal abscess. Pyogenic abscesses usually have malodorous, cream-green color and gram-negative bacteria on aspiration; however, aspiration can usually be deferred and treatment empirically started if pyogenic lesions are suspected on imaging.

Radiographic findings of hepatic abscesses include plain radiography which is usually not sensitive but can detect pneumobilia and right sided pleural effusions and ultrasound with contrast which can indicate wall-enhancement during arterial phase w/ washout in portal venous phase and internal septations in the absence of central perfusion (Figure 6). As a general rule, bacterial and fungal abscesses are often multiple, whereas amoebic abscesses are more frequently single. Amoebic abscesses are more common in a sub-diaphragmatic location and are more likely to spread through the diaphragm and into the chest. When the infection spreads to the liver through the portal veins it arises more commonly in the right lobe, probably due to an unequal distribution of superior and inferior mesenteric vein contents within the portal venous distribution. CT findings include peripherally enhancing, centrally hypoattenuating lesion as well as a double-target sign (Figure 7). The double target sign on imaging is a characteristic imaging feature of hepatic abscess demonstrated on contrast-enhanced CT scans, in which a central low attenuation lesion (fluid-filled inner ring) is surrounded by a high attenuation inner rim (abscess membrane) and a low attenuation outer ring (edema of liver parenchyma). MRI findings include hypointense central image (T1) with hyperintense perilesional edema (T2) (Figure 8). DWI (Diffusion weighted imaging): tends to have high signal within the abscess cavity 9, and high signal at the periphery ADC (apparent diffusion coefficient): tends to have low signal within the abscess cavity 9, and high signal at the periphery.

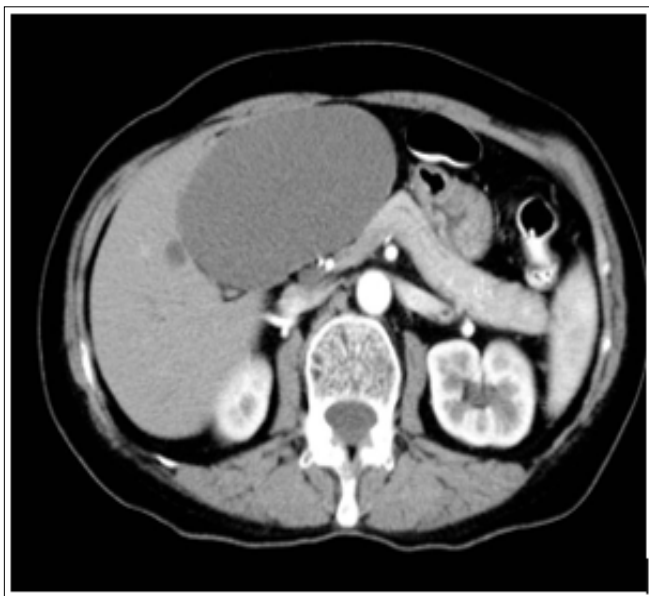


Figure 4

Diagram 1:

	AMEBIC	PYOGENIC
M:F	19:1	1.4:1
Age	28 years	44 years
Race	Hispanic (98%)	No predisposition
Birthplace	Latin America (95%)	No predisposition
RUQ pain	60%	25%
Sx > 14d	14%	40%
Cause	Entamoeba Histolytica	Biliary tract/ Diverticular Appendiceal

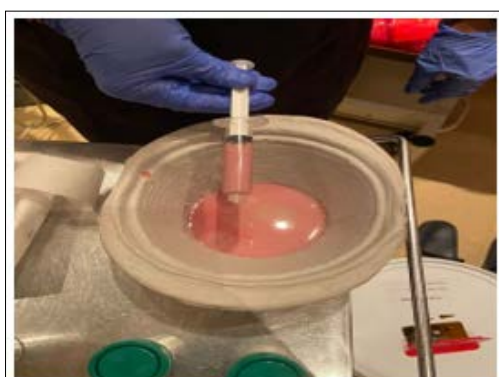


Figure 5: Amebic Abscess Drainage: Anchovy Past

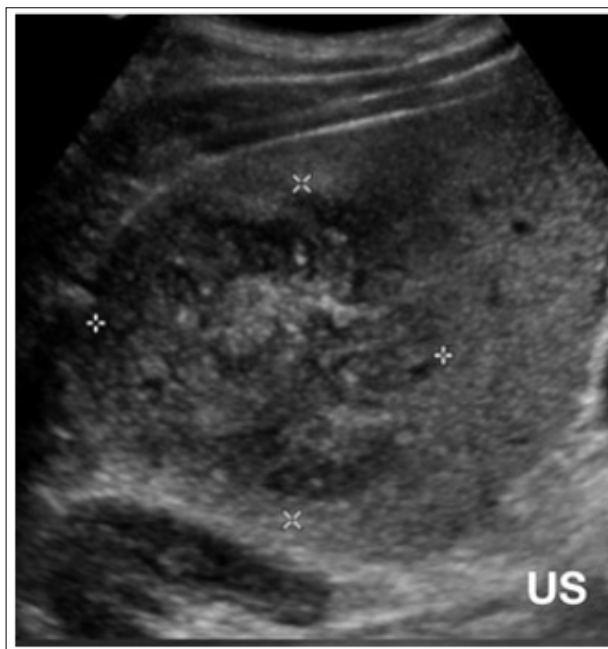


Figure 6

US



Figure 7

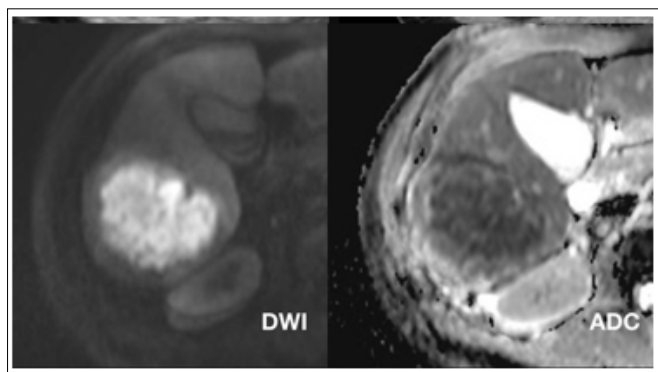


Figure 8

Treatment for hepatic abscess differs between amebic and pyogenic abscesses. Amebic abscess is treated with chloroquine for 21 days or Metronidazole for 10 days and catheter drainage is usually not needed. Pyogenic liver abscesses are treated with antibiotics based on the culture sensitivity and catheter drainage varies and is controversial since prior studies have demonstrated good antibiotic penetrance and improvement without drainage. Small abscesses (under 3 cm) and in highly septate abscesses, drainage is not recommended.

Another category of hepatic lesions including hepatic tumors that can be subdivided into malignant and benign. Malignant hepatic lesions include hepatocellular carcinoma (HCC). HCC is the 5th most common cancer in the world and 3rd most common cause of cancer-related death (after lung and stomach cancer). HCC has a high incidence in Africa, Southeast Asia (acute, aggressive), occurring in 3-5th decade, male predominance (6:1) and occurring in cirrhosis patients (65%) with hepatitis B infection being an important risk factor. HCC has a low Incidence in America and Europe with a more insidious and indolent process, occurring in the 5-6th decade, male predominance (2:1) and majority occurring in cirrhotic patients (85%) with alcohol use being a common risk factor. Hepatitis B is a major risk factor for HCC and has the strongest association given its integration into the host genome. The age of HBV acquisition determines age of HCC development with HBV occurring in infancy and HCC developing in teens to 20s and HBV occurring in adulthood with HCC occurring in 50-70-year-old. HCC can also develop in these patients without underlying cirrhosis. Common associated chemicals/carcinogens include Aflatoxin B1 (*Aspergillus flavus*) that occurs from improperly stored food in hot, humid climates as well as alcohol which is common in Western countries.

The majority of patients have cirrhosis prior to developing HCC with HBV (40-50%), alcohol use (5-15%) and hemochromatosis (3-27%) being the most common risk factors. Clinical presentation includes fatigue, weight-loss, dull/persistent right upper quadrant pain, sudden decompensation in a prior stable cirrhotic. Physical exam findings include hepatomegaly, hepatic bruit, and hemoperitoneum. Common lab findings include elevated AFP (80%) with AFP levels >300-500 suggestive of HCC as well as elevation in Des-γ-carboxyprothrombin which is elevated in 60-90% patients. HCC biopsy is a gold standard however imaging modalities can be diagnostic and tissue diagnosis can be deferred. MRI and CT scans are the most helpful with diagnosis of HCC. HCC radiographic findings with CT abdomen/pelvis includes arterial enhancement followed by venous washout (Figure 9). LiRADS classification is important to determine HCC diagnosis

with an importance to distinguish LiRADS 3 (intermediate probability) with 37% chance of HCC, unlikely to be HCC and LiRADS 4 which is probable (74%) chance of HCC. Treatment of HCC includes surgical resection, liver transplantation, chemotherapy, chemoradiation, alcohol injection, radiofrequency/thermal ablation, or cryotherapy with recommendations for patients to be evaluated by a multidisciplinary group. The Milan criteria which assesses liver transplantation for patients with underlying HCC include one lesion <5cm in diameter or multiple lesions that are less than 3 total lesions, <3cm in diameter and confined to one lobe without evidence of macrovascular invasion or extrahepatic spread, portal vein involvement.

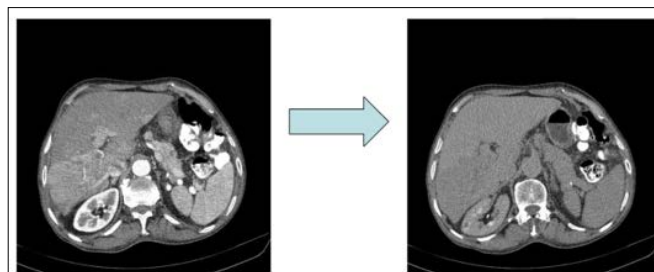


Figure 9

Venous Phase: Contrast Washes Out (venous washout) ~35sec later, Decreased Density Relative to Background Liver (“washout”)

	Arterial phase hypo- or iso-enhancement		Arterial phase non-rim hyperenhancement		
	<20 mm	≥20 mm	<10	10-19	≥20
none	LR3	LR3	LR3	LR3	LR4
one	LR3	LR4	LR4	LR4* LR5	LR5
≥two	LR4	LR4	LR4	LR5	LR5

Count additional features: Capsule - Washout - Growth

Figure 10: LiRADS Classification

Note:

LiRADS 3 (intermediate probability)=37% chance of HCC, unlikely to be HCC
 LiRADS 4 (probably)=74% chance of HCC
 LiRADS 5 (definitely)=95% chance of HCC
 -whether nodule is HCC or not (relies on arterial phase enhancement, tumor size, threshold growth over serial imaging)

Benign hepatic tumors can be subdivided into hemangioma, FNH, or adenoma. Hemangioma are defined by vascular tumors, mesenchymal origin, and blood-filled space surrounded by thin fibrous stroma which produces multiple cavernous spaces. They are mostly benign (5-7%), female predominance. Hepatic hemangioma size <4cm are incidentaloma and asymptomatic while hemangioma size >4cm are classified as giant hemangioma and are symptomatic. Hemangioma radiographic findings include CT Abdomen/Pelvis showing a hypodense lesion with irregular borders and a slow peripheral enhancement of dye coming in centrally in the tumor during the venous phase. (Figure 11), ultrasound with hyperechoic, well-defined mass (Figure 12), MRI which is very

sensitive and includes a “Light-bulb sign” on T2 (Figure 13), and a Tc99 tagged RBC scan which shows slow pooling from the periphery with time. For hepatic hemangioma biopsy is not needed and MRI scans are the most sensitive. Hemangioma treatment includes resection if >10cm in size, discontinuing estrogenic hormones and follow up imaging in 3 to 6 months. Pregnancy is discouraged if the hemangioma is large and unresectable given risk of bleeding or rupture.

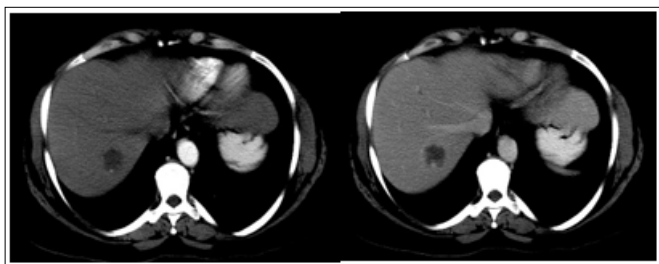


Figure 11: CT AP Hemangioma-Radiographic Findings



Figure 12

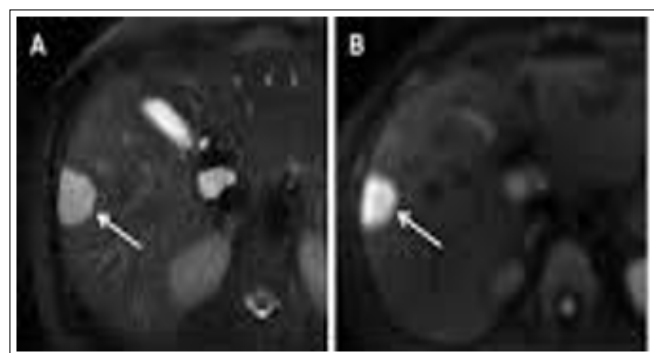


Figure 13: Light-bulb sign on MRI

Other benign hepatic tumors include adenoma and FNH. FNH is more common than adenoma occurring in all ages/both genders with peak age around 20-30s, female predominance (1:2), and are usually asymptomatic mass in the right upper quadrant with risk of rupture being rare. Adenomas commonly occur in women of child-bearing age and long-term OCP users. They are usually asymptomatic/incidental and can rupture causing hemoperitoneum with an associated high mortality (31%) and high risk of rupture during pregnancy and menstruation. Common risk factors for adenomas include oral contraceptive use (especially the first-generation pills which have a high concentration of estrogens), anabolic steroids typically in young men, obesity, metabolic

syndrome, diabetes mellitus, and glycogen storage diseases including Type I (von Gierke disease) and Type III (Cori or Forbes disease). Diagnosis for FNH is with imaging with liver tests and AFP being normal. Imaging modalities include a liver/spleen scan which can show no filling defect, an US, CT, or MRI which can show “Stellate scar” on T2 (Figure 14), and Arteriography which can show tortuous vessels w/ septations during capillary phase. For FNH, monitoring is usually not needed. Similar to FNH, adenoma diagnosis is also made on imaging since liver tests and AFP are usually normal with FNA being rarely diagnostic. Imaging can include a Liver/spleen showing a filling defect and Arteriography with spoke-wheel appearance. Monitoring is usually recommended for lesions <5cm with interval being every 6mo for two years followed by monitoring every year. If the adenoma size is greater than 5cm then more aggressive management with resection/embolization is needed. Treatment for FNH and adenoma is discontinuing OCP and surgical resection if the patient has associated complications especially in large adenomas given risk of rupture. For adenomas that are unresectable, hepatic artery ligation can be considered [1-4].



Figure 14: MRI FNH

Note: Stellate scar on T2
Avascular central stellate scar (can call it FNH)

Conclusion

Benign tumors of the liver include Liver cysts, hepatic Hemangiomas, focal nodular hyperplasia “FNH”, hepatic adenomas, and inflammatory masses of the liver.

Hepatic hemangiomas are the most common benign mesenchymal tumors of the liver occurring in up to 10% of individuals. Hemangiomas are cavernous vascular spaces lined by a single layer of flat endothelium and filled with blood with compartments separated by thin fibrous septae. Most hemangiomas do not require therapy (even if larger than 50 mm) unless symptoms or high output heart failure or Kasabach-Merritt syndrome (consumptive coagulopathy) are present. MRI is the test of choice with characteristic “centripetal enhancement” or filling of contrast from the outside in. Size alone is not an indication for surgery.

Of the malignant Liver lesions, metastatic tumors are most common, however Cholangiocarcinoma and HCC are always a concern. Hepatocellular carcinoma “HCC” is always on top of the list particularly in a background of cirrhosis. Surveillance for HCC in a patient with cirrhosis using ultrasound “US” with alpha-feto-protein has been advocated to detect earlier stage HCC. After

identifying a suspicious mass on surveillance ultrasound, cross sectional imaging study should be performed. A “triple-phase” contrasted CT or MRI is most commonly used for this purpose, and is highly accurate in inferring a diagnosis of HCC if the following criteria are met: HCC derive their blood supply from the hepatic artery “HA”, therefore HCC enhance on the arterial phase; conversely, since the HCC do not derive their blood supply from the portal vein, as does the remainder of the liver, HCC appear dark on portal venous phase (so-called “early wash-out”). In the presence of a 20 mm lesion in a cirrhotic liver with arterial enhancement with portal venous wash out on Ct or MRI, the lesion meets criteria for HCC. Masses not meeting these criteria should be biopsied, assuming safety issues have been addressed by the multidisciplinary team approach.

In short; Hepatic lesions are commonly encountered in patients and it is important to categorize the lesions in order to better manage and treat such patients. It is critical that proper assessment and

diagnosis is made for the particular liver mass, because in clinical medicine, the two drivers of any therapy are symptomatology and the malignant potential; if the patient is asymptomatic then leave them alone; “you cannot improve an asymptomatic patient” and the malignant potential, as you should not miss cancer.

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