

Diagnostic Imaging of Liver Cirrhosis in a Shih-Tzu Dog

Ho-Jung Choi, Ki-Ja Lee*, Jin-Hwa Chang, Ji-Young An, I-Se O, Se-Joon Ahn, Seong-Mok Jeong,
Seong-Jun Park, Sung-Whan Cho and Young-Won Lee¹

*College of Veterinary Medicine · Research Institute of Veterinary Medicine, Chungnam National University,
Daejeon 305-764, Korea,*

**Department of Clinical Veterinary Science, Obihiro University of Agriculture and Veterinary Medicine, Obihiro,
Hokkaido 080-8555, Japan*

(Accepted: August 19, 2009)

Abstract : A 5-year-old, intact female Shih-Tzu dog was presented with 1 year history of icterus, ascites and anorexia. The serum biochemistry revealed elevated liver enzyme levels. Microhepatica and decreased serosal detail were detected in abdominal radiography. Abdominal ultrasonographic findings included irregular liver margins, multifocal hypoechoic nodules in the liver parenchyma, and ascites. Computed tomography (CT) showed multifocal hypodense nodules with ring-like contrast enhancement. Cytologic and histopathologic examination by liver core biopsy revealed fibrosis. Cirrhosis was diagnosed based on above results. This report focuses on the imaging characteristics of ultrasonography and CT for liver cirrhosis in a dog.

Key words : cirrhosis, computed tomography, dog, ultrasonography.

Introduction

Liver cirrhosis is the end-stage of chronic hepatitis and is defined as a diffuse process characterized by fibrosis of the liver and the conversion of normal hepatic architecture into structurally abnormal nodules, and the presence of portal-central vascular anastomosis (2,21-25). Cirrhosis is a rather condition in dogs, however, the etiology of cirrhosis has been poorly understood (18,19,21,23,25). Most common clinical signs associated with cirrhosis are depression, ascites, vomiting, weight loss, anorexia, and jaundice (3,21). The definitive diagnosis is made only through the histopathology (16). In addition, other investigative modalities for the diagnosis of cirrhosis are clinical features, clinicopathologic findings, and diagnostic imaging (7,16,17,20,21). Unfortunately, all these modalities lack sensitivity and specificity and could detect patients with cirrhosis at an advanced stage (16). As imaging modalities have been unpredictably developed, however, imaging diagnoses provide accurate detection and characterization of lesions and higher sensitivity compared to the previous ones (16,21). There have been little reports for diagnostic imaging of cirrhosis in the veterinary medicine. This report describes the ultrasonographic and CT features of cirrhosis in a Shih-Tzu dog and suggests advantages of these diagnostic methods for the evaluation of cirrhosis.

Case

A five-year-old, intact female Shih-Tzu dog was presented with 1 year history of icterus, ascites and anorexia. The complete blood analysis was within reference range. The serum biochemical profiles revealed elevated liver enzymes (ALT: 137 IU/L, AST: 84 IU/L, ALP: 434 IU/L, GGT: 55 IU/L), hyperbilirubinemia (1.7 mg/dl) and hypoalbuminemia (2.4 g/dl). Abdominal radiography showed microhepatica and mildly decreased serosal detail (Fig 1). Abdominal ultrasonography revealed multifocal hypoechoic nodules in the liver parenchyma and a small amount of free fluid in the abdominal cavity (Fig 2). Ascites was disclosed transudate via ultrasound-guided abdominocentesis. Bilirubinuria and proteinuria were detected in urinalysis. The result of polymerase chain reaction (PCR) for canine adenovirus type 1 (CAV-1) using blood sample was negative. An abdominal CT (CT Max[®], GE, USA) in transverse plane using soft tissue window was performed with a thickness of 5 mm (with an absence of interslice gap). Diffuse heterogeneous parenchyma with mildly lobulated liver margins was observed (Fig 3). Multifocal hypodense nodules with ring contrast enhancement in the liver were notably seen on portovenous phase (Fig 3). The histopathologic finding of the specimens revealed infiltration of fibroid cells around the necrotic hepatocytes through US-guided core biopsy of the liver (Fig 4). Taken together, it was diagnosed as the liver cirrhosis.

Treatment was supportive and symptomatic therapy through

¹Corresponding author.
E-mail : lywon@cnu.ac.kr

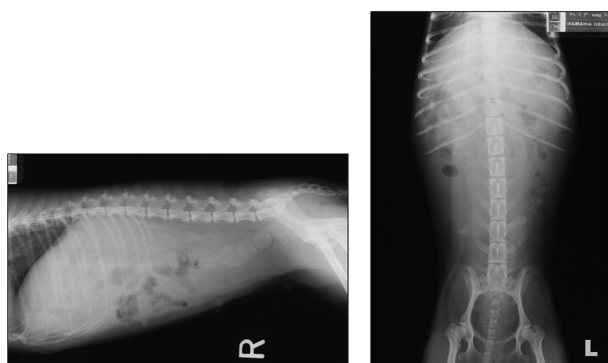


Fig 1. Lateral and ventrodorsal radiographs of the abdomen. There is mild cranial displacement of the gastric axis suggesting microhepatica. The abdominal detail is mildly decreased due to ascites.

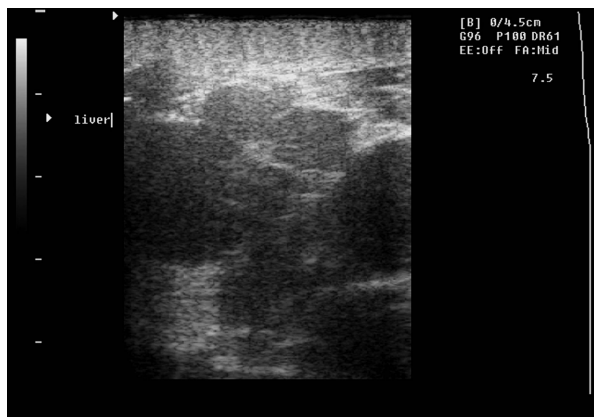


Fig 2. Multifocal hypoechoic nodules are found throughout the hepatic parenchyma. Increased echogenicity and a coarse echo-pattern of surrounding liver parenchyma are detected.

diuretics, antioxidants, liver protectants, and steroids. Follow-up examination was monitored with CBC, serum biochemistry, radiography, and ultrasonography with 4 weeks intervals.

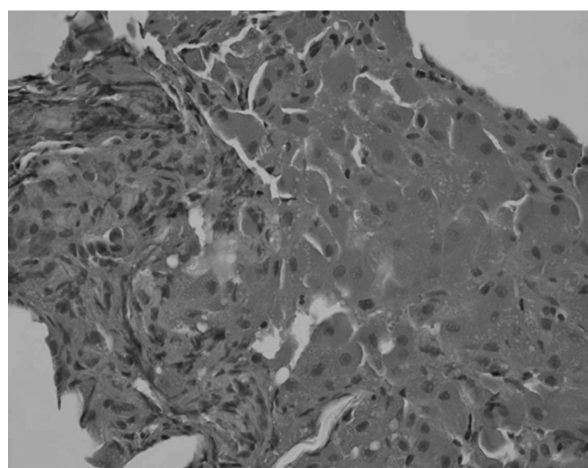


Fig 4. The histopathologic finding of the liver reveals infiltration of fibroid cells around the necrotic hepatocytes.

CT recheck was performed and has showed little change in 3 months later. The coagulopathy was identified at that time. Hypoechoic nodules in the liver were changed into the anechoic nodules after 6 months in abdominal ultrasonography (Fig 5-A). The liver parenchyma became also more heterogeneous, and the hepatic margin became more irregular and round (Fig 5-B). The patient gradually got worse by presenting weight loss, increased ascites, and prominent reduced hepatic volume for 8 months. The patient survived for 10 months after the time of evaluation.

Discussion

The liver aids greatly in the maintenance of metabolic homeostasis, detoxification, producing clotting factors and bile, and storing glycogen (6,8). The liver has a remarkable regenerative capability and cirrhosis is mostly an indolent disease (6,13,23). In humans, etiology of liver cirrhosis is well identified and most common causes are alcohol, chronic

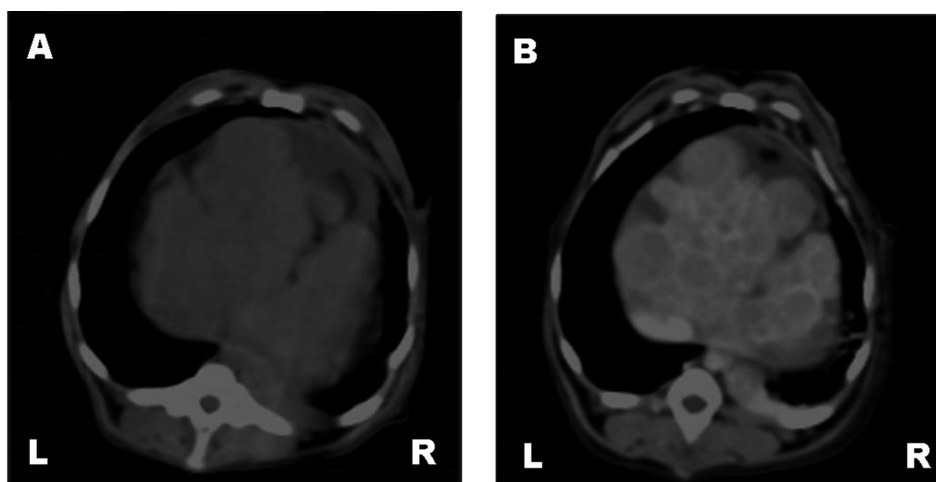


Fig 3. Transverse precontrast (A) and postcontrast (B) CT images. Noncontrast CT shows mild lobulation of hepatic contour and a diffuse heterogeneous density (A). Multiple hypodense nodules with ring shaped contrast enhancing in the liver are seen.

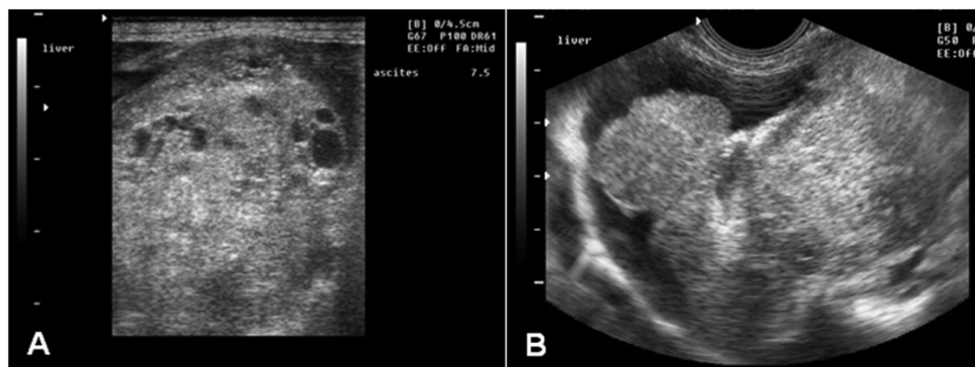


Fig 5. Ultrasonograms 6 months after Fig 2, anechoic multiple nodules of various sizes are distributed in the hyper-echoic liver (A). The blunted, irregular margin of liver was visualized because of the presence of anechoic peritoneal effusion. (B).

hepatitis, obesity, biliary obstruction, drug, and toxins (6). Only a few reports suggest relationship between the copper toxicosis and chronic hepatitis, and cirrhosis in some breeds of dogs (9,14). Some of the etiologies of hepatic damage in dogs are direct toxic effect, anoxic injury from disturbances in intrahepatic circulation, hepatocellular damage, and cholestatic liver damage (21). Chronic hepatitis can also progress to cirrhosis (3,19).

Clinical signs of cirrhotic patient are often initially vague and nonspecific, such as anorexia, lethargy, depression, weight loss, vomiting, diarrhea, polyuria, and polydipsia (11,18,21). With increased severity of hepatic dysfunction, signs of overt liver failure develop, such as ascites, jaundice, and hepatic encephalopathy (11,18,21). This patient presents with typical clinical features of hepatic disease including ascites and icterus.

Enzyme tests that are available to evaluate the liver disease are ALT, ALP, GGT, and AST (17,21). Especially, ALT and ALP are the most accepted enzymes used in small animals (21). Previous studies suggest that there is no correlation between biochemical parameters and the severity of morphologic changes or survival time in the cases of chronic hepatitis (4,18,21). Cirrhosis results in intrahepatic cholestasis causing elevated bilirubin levels; therefore, patients with cirrhosis will show a mild elevation in serum bilirubin. Low blood urea nitrogen (BUN) levels, elevated blood ammonia values, hypoalbuminemia, and abnormal clotting time result from abnormal hepatic function (21). In particular, bleeding abnormalities with cirrhosis signify a very poor prognosis (21). The characteristic clinicopathologic features such as elevated liver enzymes, hypoalbuminemia, and hyperbilirubinemia were detected in this case. Finally, the patient died after 7 months since the bleeding disorder was detected.

Abdominal radiography, ultrasonography, CT, and magnetic resonance imaging (MRI) have been used for evaluation of morphologic characteristics of cirrhosis in human medicine (6,22). Abdominal radiographs are unremarkable except when advanced stages of disease are accompanied by microhepatoma or ascites, therefore this examination is not considered as a diagnostic standard (6,11,21). Microhepatoma is usually

observed with hepatic fibrosis, atrophy, or cirrhosis, because parenchymal tissue is replaced by fibrous tissue (11,21).

Ultrasonographic hallmarks of cirrhosis are nodular and irregular liver margins, small liver size, increased hepatic echogenicity, focal lesions representing regenerative nodules and ascites (1,6,15). The detection of hepatic nodules sometimes requires further evaluation, because tumor such as hepatocellular tumor (HCT) could have similar appearances (6). Furthermore, ultrasound is an essential and non-invasive tool for the management and monitoring of cirrhotic patients. It was possible to predict poor prognosis of this patient through findings such as increased ascites, reduced liver size, progressive heterogeneous distribution of liver parenchyma, and degenerative cyst formations (1).

Typical cirrhotic morphologies on CT show heterogeneity of liver parenchyma, surface nodularity, caudate lobe hypertrophy, segmental hypertrophy and atrophy, patchy fibrosis or lace-like pattern of fibrosis, regenerative nodules in human (2,6). CT can also accurately evaluate detail distortion of hepatic architecture, general distribution and change of hepatic lesions, the degree of ascites, and varies in advanced disease (5,6). In cirrhosis, two morphological categories can be distinguished including micronodular and macronodular cirrhosis, which could be well detect on CT and MRI rather than ultrasound (21,22,25). Macronodular cirrhosis is characterized as large irregular nodules greater than 3 mm with different size (21,25). Macronodular cirrhosis is most commonly associated with chronic viral hepatitis or hepatocellular carcinoma (10,22). It is also very important to differentiate cirrhosis from hepatocellular carcinoma because both regenerative and dysplastic nodules may mimic premalignant dysplastic nodules of hepatocellular carcinoma (2,5,22). Hepatocellular carcinoma usually presents heterogeneous, moderately enhancing lesion during arterial phase, however, cirrhotic lesions show delayed, persistent contrast enhancement on portovenous phase due to the retention of contrast by the fibrotic tissue (5,22).

Although many human reports described characteristics of cirrhosis on CT images, CT examinations of the liver in dogs and cats is mainly performed for the identification of hepatic shunts and other vascular anomalies. Here, we reports the

images of liver cirrhosis on abdominal CT, and evaluates characteristic appearances, macronodular change of liver parenchyma, distortion of hepatic architecture, and ring-like contrast enhancement pattern in this case.

References

1. Biller DS, Kantrowitz B, Miyabayashi T. Ultrasonography of diffuse liver disease. *J Vet Intern Med* 1992; 6: 71-76.
2. Brancatelli G, Federle MP, Ambrosini R, Lagalla R, Carriero A, Midiri M, Vilgrain V. Cirrhosis: CT and MR imaging evaluation. *Eur J Radiol* 2007; 61: 57-69.
3. Center SA. Chronic hepatitis, cirrhosis, breed-specific hepatopathies, copper storage hepatopathy, suppurative hepatitis, granulomatous hepatitis, and idiopathic hepatic fibrosis. In: Strombeck's small animal gastroenterology, 3rd ed. Philadelphia: W.B.Saunders. 1996: 705-765.
4. Fuentealba C, Guest S, Haywood S, Horney B. Chronic hepatitis: a retrospective study in 34 dogs. *Can Vet J* 1997; 38: 365-373.
5. Gupta AA, Kim DC, Krinsky GA, Lee VS. CT and MRI of cirrhosis and its mimics. *Am J Roentgenol* 2004; 183: 1595-1601.
6. Heidelbaugh JJ, Bruderly M. Cirrhosis and chronic liver failure: part 1. Diagnosis and evaluation. *Am Fam Physician* 2006; 74: 756-762.
7. Hendrix AD. Diagnosis of chronic active hepatitis in a miniature schnauzer. *Can Vet J* 2004; 45: 765-767.
8. Herdt T. Postabsorptive nutrient utilization. In: Textbook of veterinary physiology, 3rd ed. Philadelphia: W.B. Saunders. 2002: 304-322.
9. Hoffmann G, van den Ingh TS, Bode P, Rothuizen J. Copper-associated chronic hepatitis in Labrador Retrievers. *J Vet Intern Med* 2006; 20: 856-861.
10. Ito K, Mitchell DG, Hann HW, Kim Y, Fujita T, Okazaki H. Viral induced cirrhosis: grading of severity using MR imaging. *Am J Roentgenol* 1999; 173: 591-596.
11. Johnson SE. Chronic hepatic disorders. In: Textbook of veterinary internal medicine, 5th ed. Philadelphia: Saunders. 2000: 1298-1325.
12. Larson MM. The liver and spleen. In: Textbook of veterinary diagnostic radiology, 5th ed. St. Louis, Saunders. 2007: 667-692.
13. Leveille CR, Arias IM. Pathophysiology and pharmacologic modulation of hepatic fibrosis. *J Vet Intern Med* 1993; 7: 73-84.
14. Mandigers PJ, van den Ingh TS, Spee B, Penning LC, Bode P, Rothuizen J. Chronic hepatitis in Doberman pinschers. *Vet Q* 2004; 26: 98-106.
15. Nyland TG, Mattoon JS, Herrgesell EJ, Wisner ER. Liver. In: Small animal diagnostic ultrasound, 2nd ed. Philadelphia: W.B. Saunders. 2002: 93-127.
16. Ong TZ, Tan HJ. Ultrasonography is not reliable in diagnosing liver cirrhosis in clinical practice. *Singapore Med J* 2003; 44: 293-295.
17. Sevelius E. Diagnosis and prognosis of chronic hepatitis and cirrhosis in dogs. *J Small Anim Pract* 1995; 36: 521-528.
18. Shih JL, Keating JH, Freeman LM, Webster CR. Chronic hepatitis in Labrador Retrievers: clinical presentation and prognostic factors. *J Vet Intern Med* 2007; 21: 33-39.
19. Sterczar A, Gaal T, Perge E, Rothuizen J. Chronic hepatitis in the dog-a review. *Vet Q* 2001; 23: 148-152.
20. Stockhaus C, Van Den Ingh T, Rothuizen J, Teske E. A multistep approach in the cytologic evaluation of liver biopsy samples of dogs with hepatic diseases. *Vet Pathol* 2004; 41: 461-470.
21. Twedt DC. Cirrhosis: a consequence of chronic liver disease. *Vet Clin North Am Small Anim Pract* 1985; 15: 151-176.
22. Ward J, Robinson PJ. How to detect hepatocellular carcinoma in cirrhosis. *Eur Radiol* 2002; 12: 2258-2272.
23. Watson PJ. Chronic hepatitis in dogs: a review of current understanding of the aetiology, progression, and treatment. *Vet J* 2004; 167: 228-241.
24. Willard MD. Inflammatory canine hepatic disease. In: Textbook of veterinary internal medicine, 6th ed. Philadelphia: Elsevier Saunders. 2005: 1442-1447.
25. Van den Ingh TS, Van Winkel T, Cullen JM, Charles JA, Desmet VJ. Morphological classification of parenchymal disorders of the canine and feline liver. In: Standards for clinical and histological diagnosis of canine and feline liver disease, 1st ed. Philadelphia: Elsevier Saunders. 2006: 85-101.

시추견에서 발생한 간경화의 영상 진단

최호정 · 이기자* · 장진화 · 안지영 · 오이세 · 안세준 · 정성목 · 박성준 · 조성환 · 이영원¹

충남대학교 수의과대학 · 동물위과학연구소, *오비히로 축산대학

요 약 : 5년령의 암컷 시추견이 일년 동안의 황달, 복수 등의 증상으로 약물 치료를 받다가 호전을 보이지 않아 내원 하였다. 혈액검사 결과 간효소 수치의 상승 및 고글로불린혈증과 저알부민혈증이 확인되었으며, 혈액을 이용한 CAV-1 PCR 검사시 음성임을 확인하였다. 일반 방사선 검사 및 복부 초음파 검사를 통해 간의 크기 감소와 간 실질에서의 저 에코성의 다발성 결절 및 복수를 관찰하였고, 복수 검사에서 누출성 복수임을 확인하였다. 복부 CT 검사에서 포도송이 모양으로 조영증강효과를 보이는 다발성 결절이 관찰되었다. 간 생검을 통한 조직병리학적 검사 결과 간 세포의 괴사와 섬유양 세포의 침윤을 확인하였다. 위의 검사 결과를 바탕으로 간경화증으로 진단하였다. 이후 정기적으로 혈액 검사, 혈청화학검사, 복부 초음파 검사 및 CT 검사를 실시했다. 내과적 보존적 치료를 실시하였고, 환자는 진단한 지 10개월 후에 사망하였다. 본 증례를 통하여 개에서 간경화증의 CT 영상의 특징을 보고하고자 한다.

주요어 : 간경화, 컴퓨터단층촬영, 개, 초음파.