

Case Report
Medical Imaging



Diagnostic imaging features of hepatic myelolipoma incarcerated in a peritoneopericardial diaphragmatic hernia in a cat

Namsoon Lee ^{1,2}, Jihye Choi ², Junghee Yoon ^{2,*}

¹Time Animal Medical Center, Daejeon 35233, Korea

²College of Veterinary Medicine and the Research Institute for Veterinary Science, Seoul National University, Seoul 08826, Korea



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*Corresponding author:

Junghee Yoon

College of Veterinary Medicine and the
Research Institute for Veterinary Science,
Seoul National University, 1 Gwanak-ro,
Gwanak-gu, Seoul 08826, Korea.

Email: heeyoon@snu.ac.kr

https://orcid.org/0000-0002-2077-763X

ABSTRACT

A 1-year-old male Persian cat was presented for castration. Liver incarcerated in a peritoneopericardial diaphragmatic hernia (PPDH) was diagnosed through pre-anesthetic tests. Multiple homogeneous hyperechoic nodules in the hepatic parenchyma were identified using ultrasound. The nodules showed decreased attenuation compared with normal hepatic parenchyma, and the herniated hepatic parenchyma showed increased arterial and decreased portal enhancement on computed tomography. From the histopathology, we diagnosed hydropic degeneration with portal fibrosis and myelolipoma. This report presents diagnostic imaging features of hepatic myelolipoma incarcerated in a PPDH in a cat. When perfusion of the hepatic parenchyma is altered, surgical treatment should be considered.

Keywords: Cat; peritoneopericardial diaphragmatic hernia; myelolipoma; diagnostic imaging

INTRODUCTION

Hepatic myelolipoma enclosed in a peritoneopericardial diaphragmatic hernia (PPDH) has been reported in cats [1-3]. It involves abnormal accumulation of adipose and hematopoietic tissues secondary to vascular and lymphatic congestion of the enclosed liver tissue with secondary chronic hypoxia, leading to an increased risk of transformation and degenerative changes [1]. As the liver is typically enclosed only for a short duration before a surgical treatment, hepatic myelolipoma has not been reported in young animals with PPDH [3]. To our knowledge, our patient is the youngest in which hepatic myelolipoma incarcerated in a PPDH has been reported. This report presents the diagnostic imaging features of hepatic myelolipoma incarcerated in a PPDH in veterinary medicine.

CASE PRESENTATION

A 1-year-old male Persian cat was presented for castration. Physical examination, routine hematology, and biochemistry test results were not remarkable. However, thoracic radiographs revealed an enlarged and globoid cardiac silhouette with a heterogeneous

ORCID iDs

Namssoon Lee

<https://orcid.org/0000-0002-9091-6957>

Jihye Choi

<https://orcid.org/0000-0002-1258-7158>

Junghee Yoon

<https://orcid.org/0000-0002-2077-763X>

Author Contribution

Conceptualization: Lee N; Data curation:

Lee N; Formal analysis: Lee N; Funding

acquisition: Yoon J; Investigation: Lee N,

Choi J; Methodology: Lee N, Yoon J; Project

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Choi J, Yoon J; Software: Lee N; Supervision:

Yoon J; Validation: Lee N, Choi J, Yoon J;

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Lee N; Writing - review & editing: Lee N, Choi

J, Yoon J.

Conflict of Interest

The authors declare no conflicts of interest.

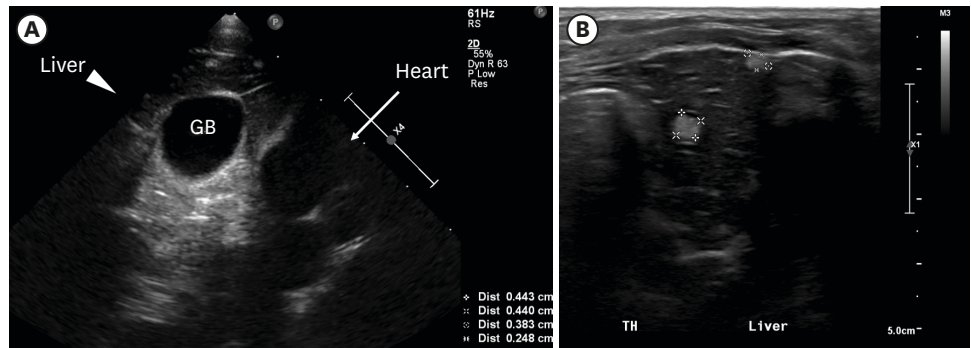


Fig. 1. Ultrasonographic images of hepatic myelolipoma incarcerated in a peritoneopericardial diaphragmatic hernia. The liver and gallbladder are within the pericardial sac (A) and multiple homogeneous hyperechoic nodules with well-defined margins (B, callipers) are observed in the herniated liver parenchyma. GB, gallbladder.

opacity, an indistinct diaphragmatic margin, and reduced lung fields. In the abdominal radiographs, microhepatica and cranial displacements of the stomach and spleen were also observed. These findings were consistent with a PPDH. Ultrasound (Affiniti 50, Philips Medical Systems, USA) examination of the thorax revealed most of the liver and gallbladder within the pericardial sac and cranial displacement of the heart. Multiple homogeneous hyperechoic nodules with a well-defined margin were identified in the herniated hepatic parenchyma on ultrasound (**Fig. 1**). The hepatic nodules were more hyperechoic than those of the spleen and even more hyperechoic than falciform fat. A differential diagnosis of the nodules included fatty nodules, benign nodular hyperplasia, and granulomas. There were no remarkable findings in the parenchyma of the abdominal liver and other abdominal organs, except for cranial position of the stomach, pancreas, and spleen.

Before surgical treatment, multi-phase computed tomography (CT) was performed using 16-slice multidetector CT (SOMATOM Scope, Siemens Healthcare, Germany; 130 kVp, 100 mAs, 2-mm slice thickness, 0.6-sec tube rotation time, and pitch of 0.6) to evaluate PPDH anatomy and nodule attenuation. The patient was positioned in sternal recumbency under general anesthesia (Ifran, Hana Pharm., Korea), and 600 mg iodine/kg iohexol (Omnipaque 300, GE Healthcare, China) was injected into the cephalic vein after pre-contrast scan by using the bolus tracking method via an autoinjector (Mallinckrodt, Liebel Flarsheim, USA). A circular bolus-tracking region of interest was placed within the aorta at the level of the abdominal liver end. The triggering threshold was set at 50 Hounsfield unit (HU). For the CT scan, the aortic phase was performed only in the caudocranial direction, and the other phases were performed in the craniocaudal direction. The scan range for multi-phase CT was from the cranial end of the herniated liver to the caudal end of the abdominal liver. The arterial, portal, and delayed venous phases were performed at 9, 29, and 60 sec from the beginning of contrast medium injection, respectively. The acquired images were reconstructed using 0.325-mm-thick slices and 0.3-mm reconstruction intervals. Attenuation values of the hepatic nodules and parenchyma were recorded without an area of major intrahepatic vessels in all CT phases. The CT showed that all left liver, quadrate, and right medial liver lobes, and gallbladder had herniated into the pericardial sac. Only the right lateral and caudal lobes were located in the abdominal cavity. The gallbladder was elongated, but the common bile duct was not dilated. There were no remarkable findings in the main branch of the intrahepatic portal and hepatic veins. The herniated left liver lobes had an irregular margin, and multiple hypo-attenuated nodules of diameter 2–5 mm were present in the overall herniated liver. There were not remarkable findings in the abdominal liver. Attenuation values of the overall

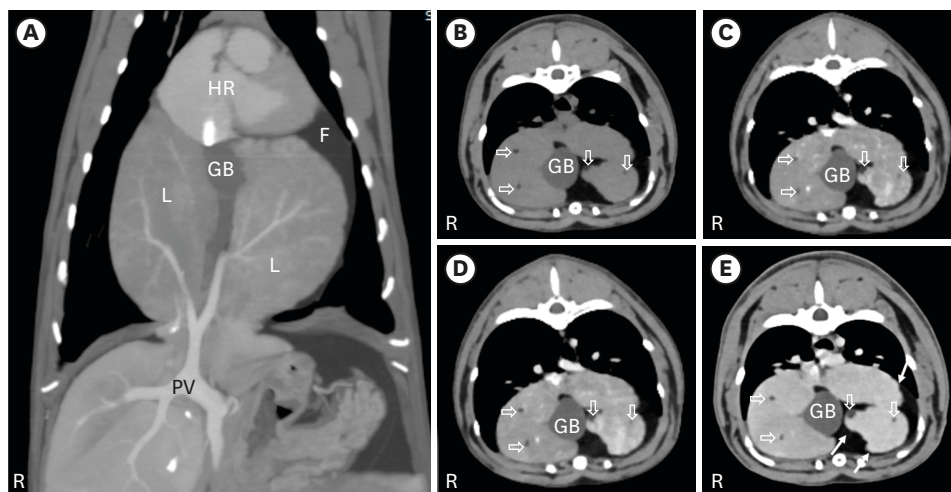


Fig. 2. Multi-phase computed tomography images of hepatic myelolipoma incarcerated in a PPDH. Dorsal thin maximum intensity projection image (A, window width 700; window level 80) of the PPDH. Transverse pre-contrast image (B), aortic phase image (C), portal phase image (D), and delayed phase image (E) of the herniated liver (window width 350; window level 50). The herniated left liver lobes have an irregular margin (white arrows), and there multiple hypoattenuated nodules (open white arrows) in the overall herniated liver. PPDH, peritoneopericardial diaphragmatic hernia; HR, heart; GB, gallbladder; L, liver; F, fat; PV, portal vein.

hepatic parenchyma was 50–60 HU on pre-contrast CT. In the aortic phase, attenuation of the abdominal liver was 70–80 HU, whereas that of the herniated liver lobe was 80–120 HU. In the portal phase, attenuation of the abdominal liver was 140–150 HU, whereas that of the herniated liver was 80–130 HU. In the delayed phase, attenuation of the abdominal liver and herniated liver was 110–140 HU. The incarcerated hepatic parenchyma showed heterogenous contrast enhancement compared with that of the normal abdominal liver parenchyma.

The attenuation value of the hepatic nodules in all the phases was 0–30 HU (**Fig. 2**). These findings were consistent with those for the hepatic fatty nodules. There were no remarkable findings in other organs except for increased attenuation of the lungs around PPDH, which was considered the compression effect of the PPDH, on CT.

A cranioventral midline celiotomy was performed to repair the PPDH. The incarcerated left liver appeared to have an abnormally gross and blunt margin and small white to yellow nodules. A biopsy of the left liver lobe was performed for histopathology. The histology revealed severe multifocal to confluent hydropic degeneration throughout the liver sample. Many lymphatics and blood vessels in the portal triad showed mild cystic dilatation. The number of small vascular channels was increased in the portal triads. Multifocal depositions of brown pigments were observed in the liver and multifocal severe fibrosis was observed in the portal triad. Multifocal fatty cells and some myeloid cells also infiltrated the hepatic parenchyma (**Fig. 3**). There was no evidence of neoplasm. Based on the histopathology results, the final diagnosis of the liver lesion was hydropic degeneration with portal fibrosis and myelolipoma. The patient recovered uneventfully and was discharged 4 d post-surgery.

DISCUSSION

This report showed the imaging features of hepatic myelolipoma. On ultrasound, fatty nodules in the liver are hyperechoic compared with those of the spleen and renal cortex [4–6]. This shows typically negative attenuation values on CT [4,6]. However, fatty lesions

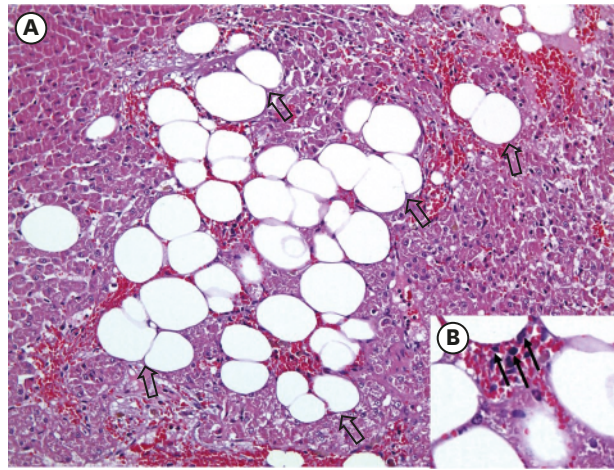


Fig. 3. Histopathology shows multifocal adipose tissues (black open arrows) and some myeloid cells (small dark basophilic cells, black arrows) infiltrated in the hepatic parenchyma. Hematoxylin and eosin staining; (A) $\times 200$ magnification; (B) $\times 400$ magnification.

do not always show negative attenuation. The non-enhanced CT attenuation of fatty lesions in the liver is lower than 40 HU or at least 10 HU lower than that of the spleen [4,5]. These lesions may show variations depending on the proportion of fat and other components [7,8]; therefore, histopathological analysis is needed to confirm fatty lesions.

In this case, hepatic parenchyma incarcerated in a PPDH showed a tendency of increased arterial enhancement, and decreased portal enhancement and heterogenous contrast enhancement compared with those of the normal abdominal liver parenchyma on multi-phase CT. In chronic liver disease, hepatic arterial perfusion increases to compensate for the decreased portal blood flow [9,10]. The present histopathology results of the liver lesion showed hydropic degeneration with portal fibrosis. Based on the histopathology results and perfusion of the herniated liver on CT, the hepatic parenchyma incarcerated in a PPDH showed chronic liver changes, although the patient had no symptoms and abnormal blood work results. In a retrospective study of dogs with PPDH, histopathologic results of the herniated liver with grossly abnormal appearance were also hepatic or portal fibrosis [11]. To our knowledge, there are no imaging studies of altered perfusion in incarcerated liver in a PPDH.

In a previous study on PPDH, long-term survival rates were similar regardless of surgical or nonsurgical treatment [12]. However, it has been reported that prognosis of and treatment choice for PPDH tend to be affected by the size of hernias and amount or type of herniated organ in dogs and cats [12]. Large defects in the diaphragm can result in adhesions of abdominal organs to the pericardium and incarcerate of organs in the pericardium [12]. Dogs that received surgical treatment tended to show clinical signs associated with PPDH, and the herniation of liver was confirmed in 66% of them [11]. It has also been reported that herniation of the liver is associated with a poor prognosis in fetal congenital diaphragmatic hernia in human medicine, suggesting compression of the lung by the herniated liver, and increased size of defect in the diaphragm and disturbed regulation of liver growth [13]. Studies have also described malignant neoplasia with or without hepatic myelolipoma in old cats with PPDH [14,15]. Thus, the prolonged enclosure of the hepatic parenchyma in a PPDH without surgical treatment can increase the risk of transformation and malignant changes because of vascular and lymphatic congestion and chronic hypoxia of hepatic tissue [14,15]. The development of myelolipomas may be associated with the presence of foci of tumor

necrosis in human medicine [16]. Therefore, as these changes are irreversible and surgical treatment of PPDH shows a good prognosis, surgical treatment should be considered in patients with hepatic myelolipoma incarcerated in a PPDH [1].

A main limitation of this report is that as it involved a single case, it was not possible to statistically prove the altered hepatic perfusion in herniated liver lobe of PPDH. Therefore, further studies with a larger number of cats are required to elucidate the hepatic perfusion in PPDH.

In conclusion, myelolipoma should be considered if hepatic parenchyma incarcerated in a PPDH has hyperechoic nodules on ultrasound and the nodules show decreased attenuation compared with normal hepatic parenchyma on CT. Furthermore, because it is suggested that perfusion of the hepatic parenchyma incarcerated in a PPDH is altered and that it may induce chronic changes in the herniated liver, surgical treatment should be considered even if the patient does not show clinical signs and abnormal blood work results.

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