

## Acquired Portosystemic Shunts Secondary to Hepatic Microvascular Dysplasia in a Young Dog

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**Abstract :** A one year old spayed female Bichon Frise dog presented with gait abnormalities and seizure. Serum biochemical results showed elevated levels of alkaline phosphatase, alanine aminotransferase, and ammonia. Serum bile acid level was also increased to be over 30  $\mu\text{mol/L}$  on preprandial. Urinalysis identified the presence of ammonium urate crystal. Abdominal ultrasonography and CT revealed aberrant, tortuous, and multiple small vessels connected to the caudal vena cava between left kidney and caudal vena cava. Macroscopic specific findings associated with extrahepatic congenital portosystemic shunts (PSS) or other liver diseases were not identified. Liver biopsy was performed. Histopathologic evaluation revealed hepatic lobular hypoplasia with portal arterial duplication and vascular shunts. Based on these finding, this case was diagnosed as multiple acquired PSS secondary to hepatic microvascular dysplasia (HMD) and hepatic encephalopathy. A liver biopsy is recommended to differentiate HMD from other liver diseases and to confirm HMD when a young dog has multiple acquired PSS.

**Key words :** microvascular dysplasia, acquired portosystemic shunt, CT, young, dog.

### Introduction

Hepatic microvascular dysplasia (HMD) describes intrahepatic microscopic shunting of blood within the liver (4). This microscopic malformation can occur in association with macroscopic congenital portosystemic shunt (cPSS). It may also occur as an isolated malformation (1,2,7).

Clinical signs of HMD vary significantly, including prolonged recovery from anesthesia, stunted growth, urinary difficulties associated with ammonium biurate crystalluria and urolithiasis, diarrhea, vomiting, and hepatoencephalopathy (1,2,4,7,8). Absence of other liver abnormalities such as macroscopic cPSS directly into the systemic circulation explains why some dogs with HMD are asymptomatic (6). Clinical signs are more common in dogs with other liver abnormalities or cPSS (6).

Histological findings of HMD are similar to those of other liver diseases that cause portal vein hypoperfusion such as portal vein obstruction and cPSS (1,2,4,7,8). The histopathology alone cannot distinguish between HMD and cPSS (6). Histologic features of HMD include an endothelial cell hyperplasia in the portal area, increased number of arterioles in the portal triads, dilatation of pericentral vascular spaces, and central venous mural hypertrophy (1,2,4,7,8). If these features are confirmed, abdominal radiography, abdominal ultrasonography, transcolonic scintigraphy, and computed tomography (CT) are important to identify cPSS in dogs with HMD

(1,4,6).

HMD has better long-term prognosis and less severe clinical signs than cPSS if they are managed medically (4). However, HMD rarely forms multiple acquired PSS (aPSS) from hypertension and exhibits severe clinical signs (9,10). We report a rare case of HMD in a young dog with multiple aPSS presenting hepatoencephalopathy.

### Case

A one-year-old spayed female Bichon Frise (5.3 kg of body weight) was presented with neurological problems such as gait abnormalities and seizure. On initial examination, its respiratory rate, heart rate, and temperature were all within normal limits. Frontal limbs showed decreased reflexes. Hind limbs were rigidly extended. Reduced level of consciousness was identified.

Complete blood count (CBC) and serum biochemical analysis results were: alanine aminotransferase (ALT), 183 U/L (reference range 10 to 100 U/L); alkaline phosphatase (ALP), 266 U/L (reference range 23 to 212 U/L); and ammonia ( $\text{NH}_3$ ), 142  $\mu\text{mol/L}$  (reference range, 0 to 98  $\mu\text{mol/L}$ ). Serum bile acid was increased to be over 30  $\mu\text{mol/L}$  (reference range 0.2 to 4.3  $\mu\text{mol/L}$ ) on preprandial. All other laboratory values were within reference ranges. Urinalysis identified the presence of ammonium urate crystal. In combination with serum biochemical results, a diagnosis of hepatic insufficiency was supported.

Survey abdominal radiography revealed microhepatica. Abdominal ultrasonography identified microhepatica with normal echogenicity. There were aberrant, tortuous, and mul-

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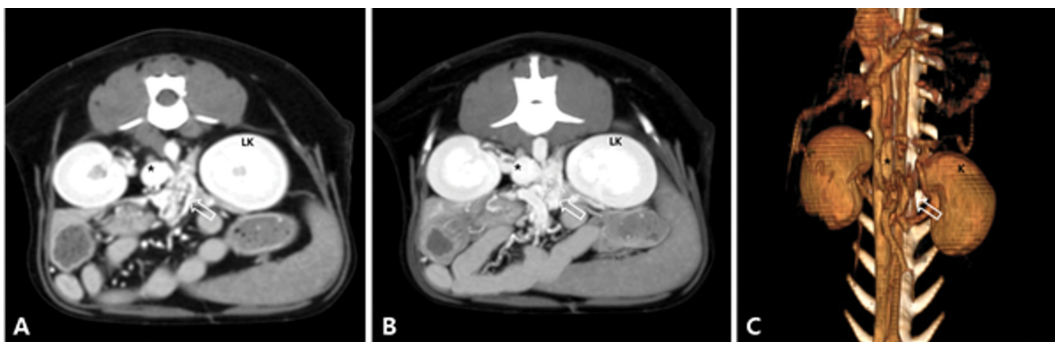
multiple small vessels between left and right kidneys. Hyperechoic crystal was identified in the urinary bladder. Other abdominal organs including intestines, pancreas, adrenal glands, kidneys, spleen, and gall bladder were normal. Abdominal CT was performed using a dual-channel multidetector helical CT scanner (Somatom Emotion<sup>®</sup>, Siemens, Muenchen, Germany) before and after automatic injection of iodinate contrast material. The following parameters were used: slice thickness of 3 mm, pitches of 1.3, tube current of 50 mAs, and X-ray tube voltage of 110 kV. A non-ionic contrast material (Omnipaque 300<sup>®</sup>, GE healthcare, Ireland) was administered (600 mgI/kg) at a rate of 1 mL/s using an automatic power injector (CT 9000<sup>TM</sup> ADV injector, Mallinckrodt, Germany). CT angiographic images were acquired from the start of arterial-phase at 10 seconds and portal-phase at 30 seconds after administering the contrast material. Near the left renal vein, there were multiple small and tortuous vessels connected to the caudal vena cava between the left kidney and caudal vena cava (Fig 1). Macroscopic specific findings associated with extrahepatic cPSS or other liver diseases were not identified. Based on these findings, multiple aPSS with HMD was the tentative diagnosis.

Exploratory laparotomy was performed for a liver biopsy. Multiple tortuous and small vessels were observed near the left kidney. The hepatic parenchyma was found to be normal. Three liver biopsy specimens were taken from right

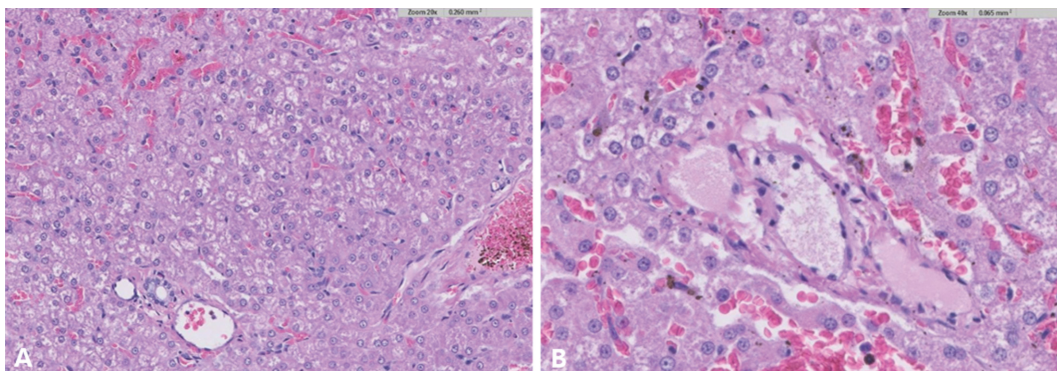
medial and left lateral lobes. Histopathologic evaluation revealed hepatic lobular hypoplasia with portal arterial duplication, vascular shunts, and multifocal mild subacute to chronic cholangiohepatitis (Fig 2), Copper stain of the liver was negative. Trichrome stain showed indentation of the capsule associated with mild capsular and subcapsular fibrosis. Based on laboratory, diagnostic imaging, and histopathologic evaluation results, this case was diagnosed as multiple aPSS secondary to HMD and hepatoencephalopathy. Recovery was uneventful. The dog was discharged three days following an exploratory laparotomy. Lactulose was administered at a dose of 1 mL per 10 kg of body weight every 8 hours. Metronidazole (15 mg/kg, PO, BID; CJ HealthCare Co., Seoul, Korea) and amoxicillin-clavulanic acid (12.5 mg/kg, PO, BID; Zoetis Pharm Co., Seoul, Korea) were continued to reduce ammonia produced by bacterial populations. Commercial therapeutic diet was also prescribed for symptomatic therapy of hepatoencephalopathy. One week after the initial presentation, serum biochemical analysis revealed that ALT, ALP, and NH<sub>3</sub> levels were 408 U/L, 163 U/L, and 27 μmol/L, respectively. No neurologic signs were found at the time of the examination at one week.

## Discussion

HMD describes histopathological vascular anomaly with



**Fig 1.** Computed tomography angiography transverse (A), maximum intensity projection transverse (B), and 3D (C) images of multiple aPSS. Near the left renal vein, multiple small and tortuous vessels (arrow) were connected to the CdVC (asterisk) between the left kidney and CdVC. aPSS, acquired portosystemic shunt; CdVC, caudal vena cava; LK, left kidney.



**Fig 2.** Histopathologic results of biopsy specimens. Histopathologic evaluation revealed multifocal mild vacuolar hepatocellular change and decreased lobule size. Mild duct hyperplasia was associated with duplicated arterioles. Histopathology showed irregular tortuous vascular shunts with asymmetrically thin and thick walls and subacute neutrophilic and chronic lymphoplasmacytic periportal inflammation associated with bile duct hyperplasia (A; H&E stain, x20, B; H&E stain, x40).

decreased blood flow in tertiary branches of the portal vein (6). This is accommodated by increased compensation for hepatic arterial perfusion (6). Histopathologic features include increased number of arterioles in the portal triads, dilatation of pericentral vascular spaces, and central venous mural hypertrophy (1,2,4,7,8). These histopathological findings overlap with all conditions that showed impaired portal vein perfusion. Especially, histopathology cannot distinguish between HMD and cPSS (6). Diagnostic imaging examinations such as abdominal ultrasonography, scintigraphy, and CT are important to rule out a macroscopic cPSS (4). Some dogs with HMD suffer from portal hypertension and develop multiple aPSS (1,10). Our case was ruled out macroscopic cPSS and revealed multiple aPSS by the ultrasonography and CT examination. Based on histopathology features and diagnostic imaging examinations, the present case was diagnosed as multiple aPSS secondary to HMD in a young dog.

Dog breeds commonly affected by HMD include Cairn Terriers and Yorkshire Terriers (4,6,8). Other affected breeds are West Highland White Terriers, Bichon Frise, Norfolk Terriers, Papillons, Pugs, Miniature Schnauzers, Shih Tzus, and Maltese (6). The average age at presentation for dogs with HMD is older than that for dogs with cPSS, which has been reported to be 6 to 18 months (5,7). CBC and serum biochemical results in HMD are generally unremarkable (4,8). Hyperammonemia is not shown in HMD without concurrent cPSS or portal atresia (6). Dogs with HMD have better long-term prognosis and less severe clinical signs than those with cPSS (4). However, HMD rarely forms multiple aPSS secondary to portal hypertension. It exhibits neurologic signs such as hepatoencephalopathy. Of 42 dogs diagnosed with multiple aPSS caused by HMD, most of these dogs were euthanized owing to disease progression or expiration (10). Our case was suspected to be portal hypertension secondary to HMD in a young Bichon Frise dog. Increased hepatic enzyme activities of ALT and ALP and hyperammonemia were identified. Elevated total serum bile acid concentration was also confirmed, indicating abnormal liver function or perfusion. Based on these findings, we expected the prognosis to be poor.

Limitation of this study was that portal vein pressure was not measured in our case. However, multiple aPSS were identified, indicating portal hypertension. Multiple aPSS can develop as a result of intrahepatic or prehepatic portal hypertension (3).

## Conclusion

This case report characterized multiple aPSS secondary to HMD in a young dog. A liver biopsy is recommended to differentiate HMD from other liver diseases and to confirm HMD when a young dog has multiple aPSS.

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