Hepatic arteriovenous fistula is a condition characterized by a communication between the portal or hepatic vein and hepatic artery. All previous case reports of dogs regarding this condition showed vascular abnormality with communication between the portal and hepatic artery [1, 3, 6, 7, 9, 13]. Dogs with hepatic arteriovenous fistula usually exhibit ascites and secondary multiple portosystemic shunt (PSS) due to portal hypertension [8, 12]. This report presents an extremely rare case of a dog with hepatic arteriovenous fistula accompanied by congenital intrahepatic PSS and aortic stenosis.

A two-month-old male golden retriever was presented to the Koide Animal Hospital, Okayama with a history of ascites which appeared suddenly two weeks before and malnutrition. The dog had been previously diagnosed with aortic stenosis accompanied by hypoalbuminemia and hyperammonemia in another hospital two weeks before and had been undergoing treatment by administration of furosemide (2 mg/kg, twice a day[bid]), spironolactone (1 mg/kg, bid), alacepril (2 mg/kg, once a day[sid]) and feeding with a protein restricted diet (Hill’s Prescription Diet l/d, Hill’s pet Nutrition, Inc., Topeka, KS, U.S.A.). A male littermate of the dog had undergone surgical correction of intrahepatic PSS previously.

Physical examination revealed that the dog was slightly emaciated, and the body weight was 5.55 kg, systolic ejection murmur over the aortic valve area and continuous murmur (bruit) with thrill through the abdominal wall over the area of the liver. However, there was no sign of ascites during examination.

Laboratory examination revealed mild microcytic (mean cell volume = 59 fl), hypochromic (mean corpuscular hemoglobin concentration = 31.5 g/dl) anemia (packed cell volume = 25%), mild prolongation of clotting time by heparplastin test (25.0 sec; normal, 13–18 sec) and activated partial thromboplastin time (26.9 sec; normal, 18–22 sec), serious panhypoproteinemia (2.5 g of protein/dl, 1.2 g of albumin/dl), high of serum alkaline phosphatase activity (1,513 U/l), slightly high fasting serum bile acid concentration (7.2 µmol/l) and blood ammonia value (137 µg/dl), and remarkable high postprandial serum bile acid concentration (133.1 µmol/dl) and blood ammonia value (261 µg/dl). The urine contained a lot of uric acid crystals.

Thoracic radiographs showed projection of aortic arch and mild right ventricular enlargement. Subaortic stenosis was confirmed through echocardiography. Abdominal radiographs revealed a slightly small liver. Ultrasonography of the liver showed two anechoic elliptical cysts which were distinct from the gallbladder within the right hepatic lobe. Color Doppler and pulsed wave Doppler ultrasound examinations demonstrated pulsatile Doppler pattern in the blood flow with systolic peaks in the cysts (Fig. 1). Cardiac...
catheter examination revealed subaortic stenosis with bronchoaortic constriction rear expansion (Fig. 2a). The gradient between aortic pressure and left ventricular pressure during the contraction phase was 21 mmHg. Celiac angiography showed hepatic arteriovenous fistula (Fig. 2b, c).

Prior to surgery, the dog was fed with a protein restricted diet (Waltham’s Canine Hepatic Support Diet, Effem GmbH, Germany) and given amoxycillin (10 mg/kg, bid), furosemide (2 mg/kg, bid), spironolactone (1 mg/kg, bid) and enalapril maleate (0.5 mg/kg, sid). The dog was premedicated with glycopyrrolate (0.02 mg/kg, subcutaneously), midazolam (0.5 mg/kg, subcutaneously) and buprenorphine HCl (0.04 mg/kg, intramuscularly). Anesthesia was induced with propofol (5 mg/kg, intravenously, bolus) and maintained using isoflurane in 100% oxygen. In addition, the dog underwent controlled ventilation under intermittent intravenous administration of vecuronium bromide. A 5Fr. angiographic catheter was inserted in the abdominal aorta via the right femoral artery and was fixed in the celiac artery. The catheter was used for celiac angiography and arterial blood pressure monitoring. The abdomen was opened through a ventral midline laparotomy. A 20 gauges intravenous canula was fixed in the mesenteric vein and used for portography and monitoring of portal blood pressure. Determination of portal blood pressure showed 8 mmHg, which was within the normal range. Mesenteric portography showed intrahepatic PSS of the central-divi-sional shunt type (Fig. 2d, e)[4]. Upon opening of the abdominal cavity, the liver was observed to be slightly small and anomalous tortuous vessels were seen in the diaphragmatic surface and margin of the right medial lobe and the quadrate lobe (Fig. 3). In addition, pulsation of the arteries along the surface of the liver was observed. In order to
approach the liver more easily, thoracotomy was performed through a median sternotomy using electric bone saw. The thoracic aorta, vena cava, celiac artery and portal vein were separated and encircled with Teflon tape then mounted with tourniquet for blocking during emergency. The big hepatic artery branch which was connected with the hepatic arteriovenous fistula was observed between the quadrate lobe and right medial lobe in the area of the porta hepatis (Fig. 4). The hepatic artery branch was isolated using an ultrasound aspirator and ligated with 1–0 silk ligature (Fig. 4). After manipulation, portal blood pressure decreased to 5 mmHg and the aortic systolic pressure rose to 134 mmHg from 90 mmHg. Signs of hepatic arteriovenous fistula disappeared when observed through selective celiac angiography (Fig. 2f). Isolation of the intrahepatic shunt vessel was tried on the hepatic diaphragm side with an ultrasound aspirator. However, isolation of the shunt vessel was unsuccessful with difficulty. The perioperative total transfusion volume was 450 ml. The dog was given continuous intravenous infusion of dopamine and dobutamine and additional blood transfusion of 180 ml postoperatively, because the dog developed hypotension and oliguria. Recovery from anesthesia was not smooth and the dog subsequently died 15 hr postoperatively.

Hepatic arteriovenous fistula in the present study was characterized by a communication between the hepatic artery and portal vein with two consecutive expansion areas observed in the right medial lobe and quadrate lobe (Fig. 4). The intrahepatic PSS was seen as a big bypass vessel which connected the portal vein to the caudal vena cava (Fig. 4). Histopathological examination of the liver showed increasing number of hepatic arterioles and bile ductal proliferation with bridge formation-related fibrosis.

This report describes an extremely rare case of a dog having three concurrent congenital anomalies with hepatic arteriovenous fistula, intrahepatic PSS and aortic stenosis. No previous report has been made regarding the presence of both hepatic arteriovenous fistula and congenital PSS in the same dog. Hepatic arteriovenous fistula in dogs is usually accompanied by ascites and secondary PSS caused by persistent portal hypertension [1, 3, 6–8, 12, 13]. In the present case, however, the portal blood pressure was 8 mmHg, which was within normal limits, and the secondary PSS was not observed. This may be the results that the arterial blood which flowed into the portal vein from the hepatic artery can flow back into caudal vena cava smoothly without passing the liver sinusoid because of the presence of a big intrahepatic PSS. The aorta systolic pressure showed a remarkable rise to 134 mmHg from 90 mmHg by correction of the hepatic arteriovenous fistula. This change suggests that a large quantity of blood flow circulated in caudal vena cava from hepatic artery via two consecutive bypasses in the liver. This disease state resembles hepatic arteriovenous fistula involving a connection between hepatic artery and hepatic vein in humans. The circulatory abnormality by the connection between hepatic artery and hepatic vein usually cause vicarious high-output heart failure in humans [8]. Preoperatively, right ventricular enlargement and diuretic drug reactivity ascites in the present case may be results of vicarious high-output heart failure. Hepatic lobectomy is recommended as surgical treatment for hepatic arteriovenous fistula with a single hepatic lobe lesion [6, 8, 12]. However, hepatic artery ramal ligation technique was chosen in the present case because the lesion extended to multiple lobes [7, 12]. Surgical management of intrahepatic PSS was attempted by isolating the shunt vessel from the liver using an ultrasound aspirator [10, 11], but this proved to be difficult and the procedure has to be abandoned because of massive bleeding with vascular injury. The correction of intrahepatic PSS should have enforced on other days to reduce operation invasion. In addition, it appears intravascular surgical technique [2] or transvenous coil embolization technique method [5] may be better options for intrahepatic PSS of this type [4].

The present report shows that congenital portal abnormality in dogs can be manifested by concurrent PSS and hepatic arteriovenous fistula.

REFERENCES


