

## Portal Vein Aneurysm in a Dog

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(Received 25 December 2011/Accepted 14 April 2012/Published online in J-STAGE 27 April 2012)

**ABSTRACT.** Portal vein aneurysm (PVA) is a rare abnormal dilatation of the portal vein, which has not been reported in dogs. We describe the findings of ultrasound and computed tomography in a case of PVA in a young male toy poodle, with the final diagnosis established by explorative surgical observation. The dog had an aneurysmal fusiform dilatation in the extrahepatic portal vein with portal hypertension and multiple portsystemic shunts. This is the first report of canine PVA.

**KEY WORDS:** canine, computed tomography, portal vein aneurysm, ultrasonography.

doi: 10.1292/jvms.11-0575; *J. Vet. Med. Sci.* 74(9): 1191–1193, 2012

One year and 11 month-old, 8.0 kg intact male Toy poodle was admitted to the Gifu University Animal Medical Center. The dog showed clinical signs including abnormal behavior such as seizures and postprandial vomiting 3 months before the 1st medical examination. A congenital portsystemic shunt (PSS) was suspected, because of relevant clinical symptoms with blood biochemical abnormalities of urea nitrogen (7 mg/dl), bile acid (over 250  $\mu\text{mol/l}$ ) and  $\text{NH}_3$  (535  $\mu\text{g/dl}$ ).

Ultrasonography and computed tomography (CT)-angiography were performed to confirm the presence of PSS. Both imaging techniques revealed a huge dilated aneurysm of the portal vein with multiple shunts from portal vein to caudal vena cava at the cranial site of the renal veins.

Ultrasonographic examination was performed using a 4–8 MHz multifrequency sector transducer. There was an anechoic large cystic structure at the craniodorsal aspect of the abdomen (Fig. 1A). This structure was located ventral to the left kidney. Since color Doppler ultrasonography revealed a broad and linear flow spectrum within the structure (Fig. 1B), the cystic structure was suspected to be part of the portal vein. Other ultrasonographic findings included microhepatia and splenomegaly. Subsequently, contrast enhanced computed tomography was performed under general anesthesia to demonstrate whether the cystic structure was part of the main portal vein. A multidetector helical CT scanner was used to obtain serial images with 1.0-mm thickness from the diaphragm to the end of the left kidney.

Post-processing images (3-dimensional reconstruction and maximum intensity projection) were made by computer software. The anechoic cystic lesion was demonstrated to be a portal vein aneurysm (PVA) based on reconstructed three-dimensional images. The PVA was shown as a large tortuous varix (4.2 cm  $\times$  2.5 cm), which laterally branched from the cranial mesenteric vein (Fig. 2A and 2B). The PVA created a mass effect on the adjacent duodenum, caudal vena cava and main portal vein (Fig. 2A). The maximum intensity projection techniques indicated that the extrahepatic portal vein was severely dilated (Fig. 2C). The intrahepatic portal venous image was not appreciable probably due to an insufficient contrast effect by the impaired blood flow into the hepatic vein. Other findings included microhepatia and multiple portsystemic shunts located near renal veins.

In the course of an explorative upper abdominal laparotomy, a mass with 4.2-cm in diameter was found at the mid abdomen (Fig. 3A). We made a definite diagnosis this structure as PVA. An extrahepatic portal vein was also dilated and meandering. There was no surrounding inflammation and pancreatitis. The portal venous pressure measured with catheter inserted in the mesenteric vein was 11 mmHg (normal range: 6–9 mmHg), which revealed mild portal hypertension. The mesentery vein was dilated in connection with portal hypertension. Splenomegaly with dilation of the splenic vein and a small amount of ascites were also observed.

The liver biopsy samples were obtained by the guillotine method from the quadrate lobe. Histopathology revealed severe blood stasis, sinusoidal dilation, proliferation of small arteries, deposits of bile pigments, and hypoplastic portal veins (Fig. 3B and 3C).

Since the multiple portsystemic shunt and portal hypertension are not a surgical disease, but rather require medical managements, oral administration of lactulose and

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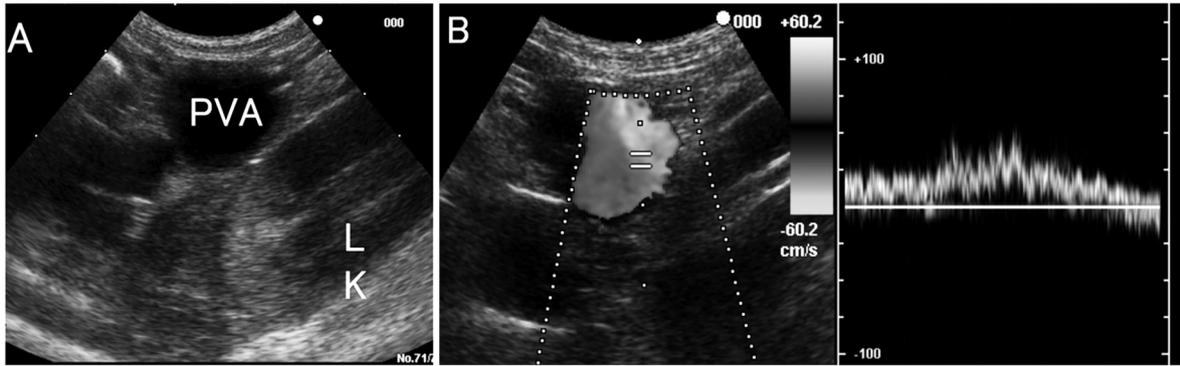


Fig. 1. (A) Transverse ultrasonographic image. A portal vein aneurysm is shown as a large cystic structure. (B) Pulsed wave Doppler in a portal vein aneurysm. Left kidney (LK), portal vein aneurysm (PVA).

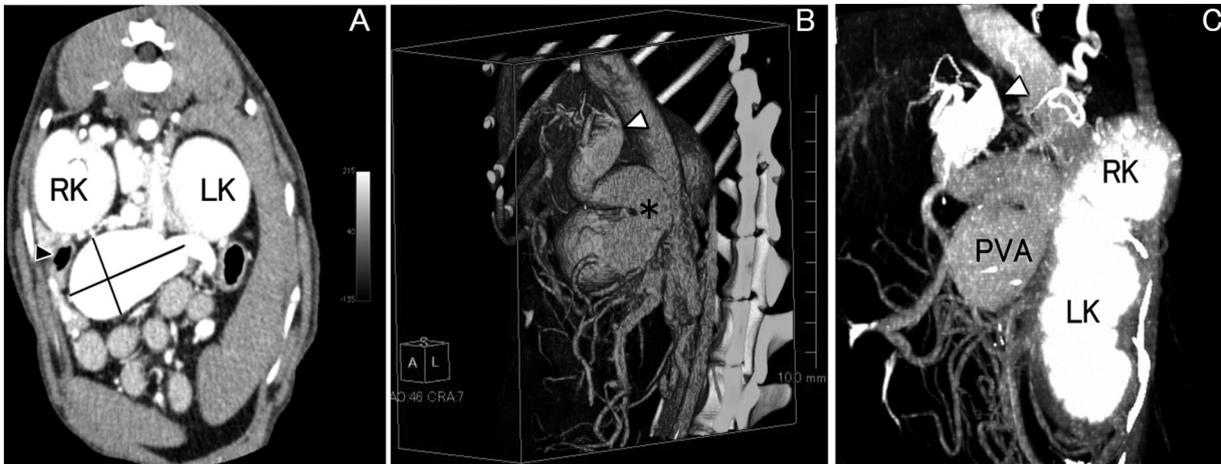


Fig. 2. (A) Transverse contrast-enhanced computed tomographic image of the abdomen at the level of the first lumbar vertebra showing the mass effect of the portal vein aneurysm on the adjacent duodenum (black arrowhead). The portal vein aneurysm measured 4.2 cm  $\times$  2.5 cm. (B) Three-dimensional computed tomographic volume rendering images were taken. The oblique view from the ventral to dorsal direction in the portal phase. Note the large portal vein aneurysm at the center of the abdomen. Portal vein aneurysm branching from the portal vein (\*\*). (C) Left lateral maximum-intensity projection with blood stagnation in portal vein. Extrahepatic portal vein (white arrowhead), Right kidney (RK), Left kidney (LK), portal vein aneurysm (PVA).

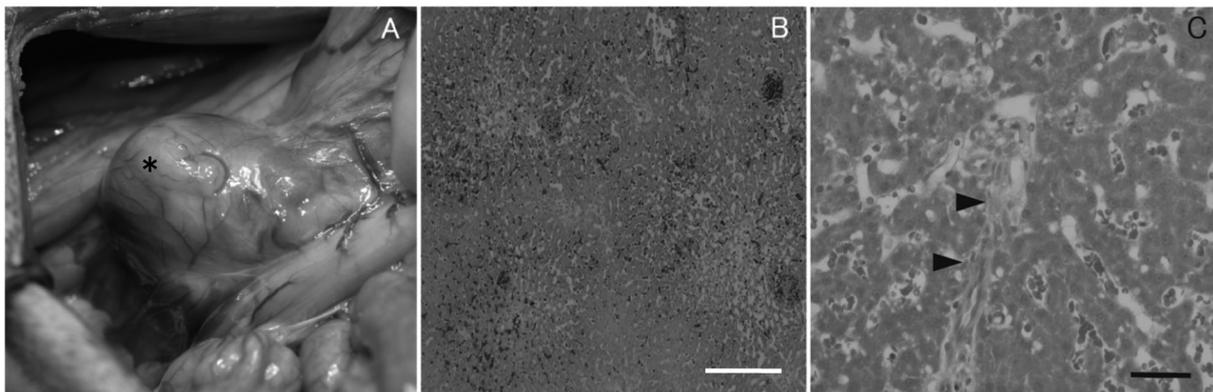


Fig. 3. (A) Gross observation of the explorative abdominal laparotomy. Fusiform structure under the duodenum is the portal vein aneurysm (\*). Upper left is cranial side. (B and C) Histopathological image of the hepatic lesion. Disappearance of the portal tract was seen at some hepatic triad. Sinusoidal dilation occurs from the central vein. Proliferation of small arteries (black arrowhead) can be seen. Hematoxylin and eosin stain. White and black scale bars indicate 400  $\mu$ m and 50  $\mu$ m.

branched-chain amino acid was administered.

Ten months after the diagnosis, the patient was still alive without any clinical symptoms.

In humans, the extrahepatic PVA is a rare vascular anomaly of the portal venous system. Since first reported in 1956 by Barzilai and Kleckner, only about 70 cases have been published to date [1, 7, 9, 11]. Human PVA is defined as a focal saccular or fusiform dilatation [1], and a segmental dilatation larger than 1.4 cm is considered to be a PVA [7]. According to the diagnostic criteria for human PVA, we diagnosed the present canine case with PVA, because a large aneurysm in a part of the main portal vein was 4.2 cm in diameter.

One case had been reported about aneurysmal portal vein with complex congenital intrahepatic portosystemic shunt in a dog [6]. This case can be differentiated from ours by the absence of several signs of portal hypertension such as multiple acquired shunts and ascites. The pathophysiology of this reported case is opposite to our case either portal hypotension or portal hypertension. A significant number of previously reported cases of portal venous system aneurysms were associated with portal hypertension [9].

Although the pathogenesis of PVA remains unclear, either congenital or acquired causes have been suspected in humans [5]. Congenital PVA has been hypothesized as results of an inherent malformation of the vitelline vein and weakness of vessel walls [7]. On the other hand, acquired PVA may be arisen from portal hypertension, necrotizing pancreatitis, abdominal trauma, or surgical iatrogenesis [5, 11]. In our case, there was a mild portal hypertension but not pancreatitis or trauma. Histological findings of the liver included severe sinusoidal dilation and an increased number of arterioles in the portal triads, suggesting that the portal hypertension might be resulted from the liver disease. While several studies have been made on portal hypertension of dogs [2, 8], there has been no report as to PVA resulted from portal hypertension. Although we did not histopathologically evaluate the portal vessel, unknown congenital abnormalities on the vessel with portal hypertension might exist in the present case. Therefore, these extrahepatic and intrahepatic vascular anomalies might cause the PVA.

Patients with PVA are usually asymptomatic, however, thrombosis, rupture, and compression of adjacent organs would be the majority of complications [9, 11, 12]. These complications depend on the location and shape of PVA [3]. Almost all patients with asymptomatic PVA need to be only followed closely without surgical treatment, because the previous report suggests PVA undergoes complete regression following several years of observation [10]. However, surgical intervention may be required for PVA that is associated with symptoms or with other complications [4, 10, 11]. In our case, surgical treatment was not performed, because one of the etiologies of the PVA would result from hepatic vascular lesion that created portal hypertension and PVA. Surgical removal of aneurysm would not help increasing

hepatic blood flow and hepatic function.

Although the clinical symptom remained stable with medical management, follow-up CT and ultrasonographic examination would be necessary to evaluate the aneurysm development and the presence of mural thrombus. If a thrombus develops, surgical removal of the aneurysm may be necessary to avoid complete occlusion of the portal vein.

In this paper, we describe the imaging and explorative surgical findings of PVA that has never been mentioned in veterinary literature. Since the clinical symptoms of the present case were similar to those of PSS, radiologists may also consider PVA as one of the differential diagnosis in patients showing portal hypertension.

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