Pulsed Doppler Ultrasonographic Evaluation of Portal Blood Flow in Dogs with Experimental Portal Vein Branch Ligation

Young-won LEE
Veterinary Medical Teaching Hospital, College of Veterinary Medicine, Seoul National University, San 56–1, Shilim-dong, Kwanak-ku, Seoul, 151–742, Korea
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ABSTRACT. Portal blood flow was measured using pulsed Doppler ultrasound in 6 dogs before and after left portal vein branch ligation. Mean portal vein blood flow velocity and mean portal vein blood flow were significantly reduced after ligation and the congestion index was increased ($p<0.01$). Pulsed Doppler ultrasound studies provide valuable physiological information which may assist the clinician with the diagnosis of canine hepatic circulatory disorders.—KEY WORDS: canine, Doppler ultrasound, portal blood flow.
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Total hepatic perfusion and the relative contributions of the hepatic arterial and portal venous systems can be altered by a variety of liver diseases. These include diffuse hepatocellular disease, neoplasia, and portovascular shunts which can markedly affect hepatic circulation [12–14].

Portal hypertension occurs when there is impedence to the normal flow of abdominal splanchnic venous blood through the portal vein, liver, and caudal vena cava to the right side of the heart [1]. Portal hypertension is clinically classified as prehepatic, hepatic, and posthepatic. Prehepatic causes of portal hypertension are uncommon in dogs and rare in cats, although congenital portal atresia, congenital portal vein stenosis, neoplasia, and abscessation are occasionally reported in the dog [1, 3, 4].

The diagnosis of prehepatic portal hypertension is difficult because of nonspecific clinical signs and ambiguity of clinicopathologic features (complete blood count, chemistry profile) [1]. However, some reports suggest that Doppler ultrasound is a useful method to diagnose portal hypertension [3, 4].

Pulsed Doppler ultrasound allows simultaneous imaging and spectral analysis of hepatic vascular flow patterns in a noninvasive manner [5, 11]. These patterns have the potential to provide both qualitative and quantitative functional information relative to a particular hepatic disease process, but have previously been studied in detail in the dog [6, 11]. Preliminary studies in man have compared Doppler blood flow measurements in normal and various disease states, and the results show promise for establishing this technique as an important clinical and research tool [2, 8, 17]. Some reports of portal hypertension in dogs indicate that Doppler ultrasonography is one of the most useful methods in the diagnosis of portal hypertension [3, 4, 7].

This investigation had two purposes; first, to assess in portal hemodynamics with Doppler ultrasound; second, to document vascular changes (velocity, blood flow, congestion index) following ligation of the left portal vein branch.

Six clinically normal, adult, mixed-breed dogs were used in this study. There were 4 females and 2 males. The mean body weight of the dogs was 11.35 ± 1.05 kg (range 10.3–12.8 kg). Physical examination, complete blood count, and serum biochemical analysis were performed on each dog. Food was withheld for 12 hr prior to all the ultrasound examinations.

Dogs were anesthetized with Zoletil® (tiletamine+zolazepam, Virbac, France, 10 mg/kg, IV). The portal vein was isolated and exposed through a midline laparotomy. The left portal vein branch was double ligated with 2–0 silk. Routine post-operative care was performed after surgery.

A pulsed Doppler ultrasound system with a 5.0 MHz sector transducer (SonoRex®, Medison Ltd. Co., Korea) was used to obtain two-dimensional, real-time ultrasound images of the liver and blood vessels. The same transducer was used to obtain Doppler information.

The transducer was placed at the right 9th to 11th intercostal spaces. The Doppler cursor was placed in the mid lumen of the portal vein. Three measurements were taken from each dog at an optimal acoustic window to the portal vein. The angle of insonation was always less than 70°C. The length of sample volume was approximately half the diameter of the vessel. Pulse repetition frequency of 3.0 kHz was used and the cross sectional area of the portal vein was calculated from the vessel diameter as measured on transverse scans at right angles to the vessel wall. The maximal velocity method was used to calculate flow volume in this study. The congestion index was calculated by dividing the portal vein area by the portal blood flow velocity [9].

Doppler ultrasound examinations were performed one week before and then after left portal vein branch ligation (3, 5, 7, 9, 11, 15, 21 days after ligation).

The One-Way Analysis of Variance was used in statistical evaluation of data.

Table 1 shows serial changes of portal blood flow velocity, portal blood flow and congestion index before and after left portal vein branch ligation. The values are the means of 18 measurements (6 dogs × 3 measurements). Mean portal vein blood flow velocity and mean portal vein blood flow were significantly reduced 3 days after ligation and thereafter ($p<0.01$). The congestion index also significantly increased 3 days after ligation and thereafter

Table 1. Serial changes of portal blood flow velocity, portal blood flow, and congestion index measurement using Doppler ultrasound in the dogs before and after left portal vein branch ligation

Day	Mean Portal Vein Blood Flow Velocity (cm/sec)	Mean Portal Vein Blood Flow (ml/min/kg)	Congestion Index (cm·sec)
Before	16.9 ± 1.9*	34.2 ± 3.5*	0.0319 ± 0.0023*
After 3	15.2 ± 0.7	33.6 ± 4.5	0.0350 ± 0.0035
5	14.0 ± 0.7	30.1 ± 1.0	0.0374 ± 0.0037
7	13.3 ± 0.9	28.5 ± 1.8	0.0383 ± 0.0032
9	13.2 ± 0.3	26.9 ± 1.4	0.0406 ± 0.0014
11	13.2 ± 0.3	26.4 ± 1.5	0.0432 ± 0.0034
15	12.9 ± 0.5	27.4 ± 0.5	0.0435 ± 0.0018
21	12.0 ± 0.3	25.0 ± 0.7	0.0450 ± 0.0005

*: All the differences between the values before and after ligation were significant.

($p<0.01$).

Total hepatic blood flow in normal dogs is 30-40 m*l*/min/kg, with the portal vein and hepatic artery contributing 70–80% and 20–30%, respectively [18]. Thus, reported portal blood flow ranges between 21–32 m*l*/min/kg. The results of this investigation using Doppler ultrasound to measure portal flow at preoperative periods (34.2 ± 3.0 m*l*/min/kg) were similar to those in invasive studies [10, 15].

The average portal flow velocity may be calculated by the maximum velocity method or the uniform insonation method [11]. The maximum velocity method uses a sample volume approximately half the vessel's diameter to obtain maximum velocity [8]. Average velocity is then 57% of maximum velocity, as reported in previous *in vitro* and *in vivo* circulation model studies [8]. With both methods, velocity must be averaged over several inspiratory/expiratory cycles because a slight increase in velocity is seen at expiration versus inspiration [11].

Average portal flow velocity was calculated using the maximum velocity method in this investigation because the uniform insonation method caused vessel wall artifact and overlaping vessel artifact. A previous study reported that normal portal flow velocity was 18.1 ± 7.6 cm/sec in ten dogs [11]. In this investigation, preoperative portal flow velocity was 16.9 ± 1.9 cm/sec. A previous report indicated that significant flow velocity errors could be introduced if large incident angles were used between the central ultrasound beam and the long axis of the vessel [5]. Therefore, measurements made with high angles (>70°C) were excluded from this study.

The congestion index of the portal vein was derived from the ratio between the cross-sectional area of the portal vein and the mean velocity of portal flow. It is significantly different in diseases which alter liver cirrhosis. In humans, the congestion index increased in chronic hepatitis, liver cirrhosis, and portal hypertention. And it may provide more informations on the clinical stage of liver disease [9, 16]. The congestion index was reported to increase in dogs with liver disease [6, 11], and in this study, its increase was demonstrated after ligation of the left portal vein branch.

Portal hypertension, defined as a persistent increase in portal vascular pressure, is caused by an increase in portal venous blood flow, an increase in resistance to portal blood flow, or both [3, 4]. The most common mechanism in the development of portal hypertension in animals is an increased resistance to blood flow along the path from the portal vein to the heart. Obstruction of flow causes distention of the capillary bed behind the obstruction, with a decrease in blood flow, venous stasis, and portal hypertension [3].

Prehepatic portal hypertension refers to the obstruction of the portal vein or its branches before it enters the liver. Prehepatic portal hypertension is caused by portal vein thrombosis, stenosis, neoplasia, or portal vein compression. Currently, the diagnosis of portal hypertension is presumptive in the dog. The assessment of a patient suspected of having portal hypertension requires careful consideration of its causes and complications [1].

With acute portal vein obstruction, portal hypertension may lead to death associated with impaired visceral perfusion, endotoxemia, and thromboembolism [1, 3, 4]. When portal vein obstruction has a more gradual or insidious development, the ensuing portal hypertension can be tolerated as a result of development of portosystemic collateral circulatory pathways [1].

Pulsed Doppler ultrasound studies provide valuable physiological information which may assist the clinician with the diagnosis, management and prognosis of canine prehepatic portal hypertension. Doppler examinations supplement the anatomical information gained with conventional two-dimensional ultrasound imaging. Doppler ultrasonography showed that portal blood flow velocity and average portal blood flow gradually decreased and the congestion index gradually increased after ligation of left portal vein branch.

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