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Portosystemic Shunt in A Puppy Poodle and Medical Treatment: Case report

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ABSTRACT

The case includes a 7-month-old puppy poodle applied to a private hospital for weakness, tremors and seizure attacks. Clinical examination findings were normal. From blood samples, biochemical parameter measurements were carried out. The values of alkaline phosphatase (ALP), alanine aminotransferase (ALT), ammonia and fasting serum bile acids were high and the blood urea nitrogen (BUN) value was low. Ultrasonographic examination, shunted vein in the liver to the vena cava caudalis, that is colour Doppler observed the extrahepatic shunt and turbulent flow in this shunted. Depending on clinical, laboratory (hemogram and biochemistry) and ultrasonographic observations, portosystemic shunt (PSS) was diagnosed and controlled one month after the treatment was recommended. The medical treatment included a hepatic formula diet (liver care), lactulose 0.5 ml/kg three times a day, metronidazole 15 mg/kg twice a day, S-adenosyl Methionine 15 mg/kg once a day, 400 international unite (IU) vitamin E once a day for 30 days. When turbulent flow is observed in the shunted vein in the liver to vena cava caudalis, ultrasound examination with color Doppler can help diagnose portosystemic shunt. After the treatment, clinical improvement was observed and clinical symptoms of hepatic encephalopathy including seizures and tremors, disappeared completely. Determination of turbulent flow with colour Doppler and decreased portal flow velocity with portal hypertension with PW-Doppler ultrasonography are important for the diagnosis of the portosystemic shunt. It was concluded that medical treatments might help before surgical treatments in portosystemic shunts.

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INTRODUCTION

Portosystemic shunt occurs mostly in cats and dogs due to congenital anomalies as a result of chronic liver diseases such as hepatic cirrhosis and hepatic fibrosis, caused by portal hypertension. Portosystemic shunts are abnormal vascular connections between the portal venous system and the systemic circulation that allow portal venous blood to penetrate directly into the systemic circulation or through the liver **(Lamb 1996; Santos *et al.*, 2019).** Shunting leads to a decreased level of portal blood; thus, the liver cannot grow sufficiently and the blood cannot be filtered sufficiently in the liver. Although portosystemic shunt is seen in other animals, it mostly occurs in dogs. Congenital PSS occurs in intrahepatic or extrahepatic forms **(Tobias and Rohrbach 2003).**

Extrahepatic congenital PSS is often reported in small breeds of dogs and cats. Intrahepatic PSS is less prevalent but, it's diagnosed more frequently in large dog breeds. **(Carvalho and Chammas, 2008).** Dogs with extrahepatic portosystemic shunts (EHPSS) might possess clinical signs before 2 years of age **(Center and Magne, 1990).** Clinical signs of an affected dog might exist in three major systems: the gastrointestinal, nervous, and urinary systems. Symptoms of dogs with PSS are loss of appetite, weight loss, vomiting, polyuria, polydipsia, tremors and seizures. In laboratory findings of a portosystemic shunt, an increase in alanine aminotransferase (ALT), aspartate aminotransferase (AST), alkaline phosphatase (ALP), serum bile acids, ammonia levels and a decrease in blood urea (BUN) are found. Increased serum bile acids and ammonia levels are highly diagnostic for PSS **(Greenhalg *et al.*, 2010; Kraun *et al.*, 2014).**

In a study, **Nyland and Fischer (1990)** performed an ultrasound in dogs with experimental cirrhosis, and they found a decreased portal blood flow velocity and increased congestion. They reported that

portal hypertension with decreased portal blood flow is consistent with an extrahepatic portosystemic shunt **(Cristopher 1996). Cristopher (1996)** reported that the sensitivity and specificity of ultrasound in detecting congenital PSS were 95% and 98%. In the same study, veins with anomalies were detected in 37 of 38 dogs. Colour Doppler and PW-doppler ultrasound examination, extrahepatic portosystemic shunt, turbulent flow presence, and decreased portal vein velocity in the shunted vessel were found. Thus, colored Doppler is useful in detecting the shunted vessel **(Cristopher 1996).** Our goal was to determine the shunt with colour and PW-Doppler ultrasonographic examination. Colour Doppler shows turbulent flow and PW-Doppler shows decreased portal flow with portal hypertension.

Case presentation

In our case, a 7-month-23-day-old Poodle male puppy was applied to a private hospital with tremors and a seizure complaint. The onset of seizures includes unusual behavior, drooling, vocalizing, and wobbliness when walking. After these symptoms, tremors started. Seizures and tremors vary in intensity from day to day. There was hyperthermia (39.3^{o}C) and tachycardia (145). Respiration rate, type, and depth were normal. Mucous membranes examinations were normal and appetite slightly decreased. Other clinical examination findings were normal. The owner had not used any medicines for the puppy before the examination. From blood samples, biochemical parameter measurements were carried out. ALP, ALT, ammonia and fasting serum bile acids were high and BUN values were low (Table 1). Fasting serum bile acids were measured with the IDEXX catalyst one Chemistry Analyzer (USA) and other parameters were measured with the fuji nx-600 chemical analyzer (Japan).

As a result of the ultrasonographic such as colour Doppler and PW-Doppler examination, the shunted vein in the liver to the vena cava caudalis, that is, the extrahepatic shunt and turbulent flow in this shunted vein was observed by colour Doppler (Figure 1-2). Portal flow velocity was 3.1 cm/s with Pulse-wave Doppler (PW-Doppler). The portosystemic shunt was diagnosed based on clinical examination, anamnesis, laboratory results, and ultrasonographic examination. The Mindray Vetus 8 (China) was used for ultrasonographic imaging. After that, the puppy has sent for computerized tomography (CT) imaging to determine the shunt in the liver (Figure 3-4-5). The medical treatment included a hepatic formula diet (Hill's L/D prescription diet, liver care), lactulose 0.5 ml/kg three times a day, metronidazole 15 mg/kg twice a day, S-adenosyl methionine 15 mg/kg once a day, 400 IU vitamin E once a day. The treatment lasted for 30 days.

Table 1: Laboratory Results of the dog with Portosystemic shunt

Parameters	Results	Reference Value (Turgut 2000)
ALP (U/L)	301	10-80
ALT (U/L)	124	10-80
T.Bilirubin (mg/dL)	0.2	0.1-0.5
GGT (U/L)	10	1-10
CRE (mg/dL)	0.44	0.4-1.4
BUN (mg/dL)	9.1	12-25
NH3 (ug/dL)	308	16-75
Serum Bile Acids (umol/L)	145	0-10
Glucose (mg/dL)	95	70-150

During the follow-up visit one month after the treatment, it was observed that the seizures and tremors, which are the symptoms of hepatic encephalopathy, disappeared completely, and the appetite level and general condition of the puppy improved. Most of the biochemical parameters of the blood samples were found to be within the reference range and a significant decrease in ammonia and bile acids levels was detected (Table 2). The symptoms were controlled after medical treatment.

Table 2: Post-treatment laboratory results of the dog with portosystemic shunt

Parameters	Results	Reference Value (Turgut 2000)
ALP (U/L)	87	10-80
ALT (U/L)	65	10-80
T. Bilirubin (mg/dL)	0.3	0.1-0.5
GGT (U/L)	8	1-10
CRE (mg/dL)	0.86	0.4-1.4
BUN (mg/dL)	12.5	12-25
NH3 (ug/dL)	111	16-75
Serum Bile Acids (umol/L)	21.8	0-10
Glucose (mg/dL)	98	70-150

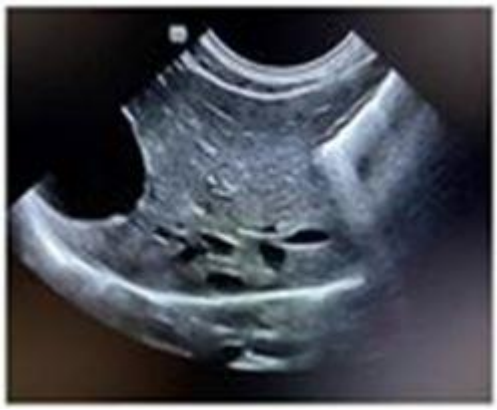

Fig. 1: Portal vein, vena cava caudalis and shunted vessels in PSS

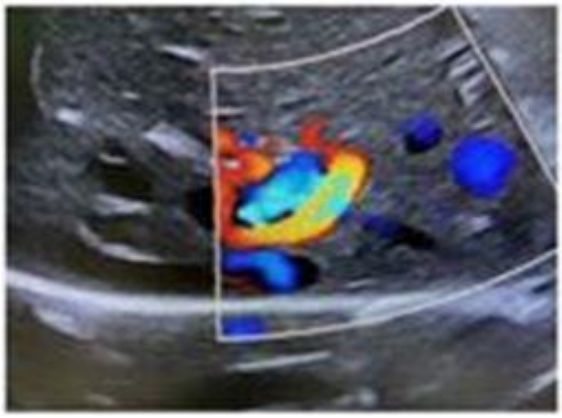

Fig. 2: Turbulent flow in the shunted vessel in PSS

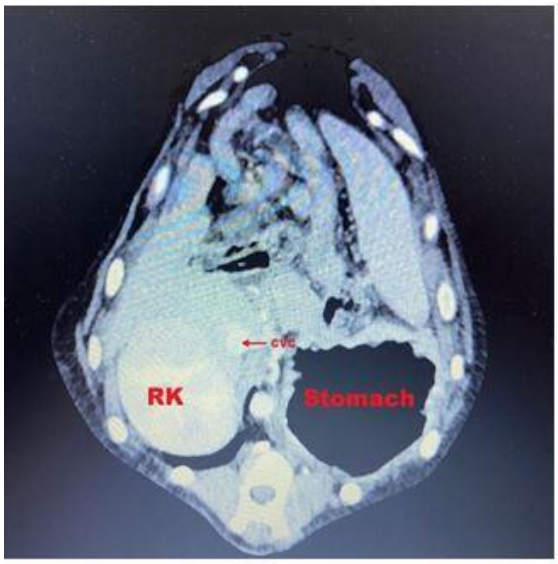

Fig.3: Contrast-enhanced computed tomographic images of the liver obtained during the portal phase (RK=Right Kidney, CVC= Cranial Vena Cava

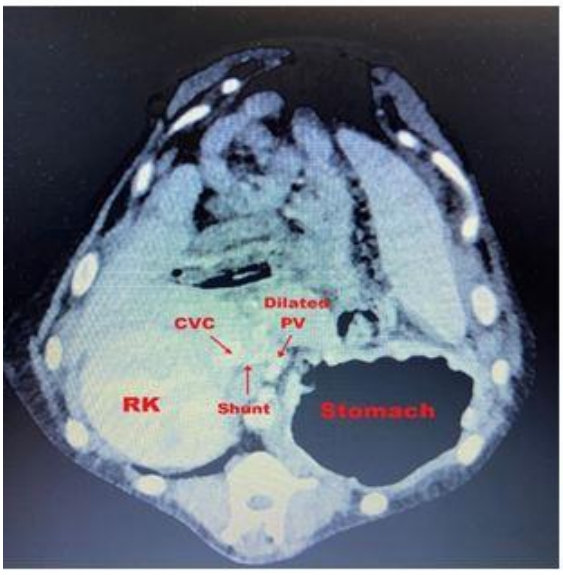

Fig.4: PSS on CT-imaging (RK=Right Kidney, CVC= Cranial Vena Cava, PV= Portal Vein.

DISCUSSION

This case report determined that ultrasonographic 2-D and colour Doppler examination has an important role in diagnosing PSS. Protein is an essential nutrient for developing and treating liver diseases in puppies. The liver's ability to metabolize amino acids and proteins is reduced in liver damage (**Butterworth *et al*., 1987; Blei, 2001)**. Decreased amino acid metabolism causes an increase in ammonia in the blood. In healthy dogs, ammonia is converted to urea by transporting the liver through the portal venous circulation (**Grant *et al.*, 2021)**. In the case of a portosystemic shunt, some ammonia is transported to the liver by portal circulation. In contrast, the rest of the ammonia enters the systemic circulation through the shunted vascular channel. This situation causes hyperammonemia. Increased ammonia in the blood crosses the blood-brain barrier and causes neurological symptoms such as ataxia, increased salivation, tremor, seizure, and lethargy (**Butterworth *et al*., 1987)**. If the puppy with PSS has these symptoms, it is actually an indication that the diet needs to be changed. Two different views on diet in PSS are recommended; total protein restriction or modification of the protein source in the diet. The liver is the organ where the degranulation of aromatic amino acids. Branch chain amino acids cannot degranulate in the liver (**Meyer *et al.*, 1999; Grant *et al*., 2021)**.

Therefore, in liver damage, branch-chain amino acids are more beneficial than aromatic amino acids as a source of protein in the diet **(Meyer *et al.*, 1999; Grant *et al.*, 2021). Grant *et al.*, (2021)** reported that there was no difference in the reduction or prevention of hepatic encephalopathy of chicken-derived or egg and soy-derived foods as a protein **(Proot *et al.*, 2009; Gtant *et al.*, 2021).** Therefore, protein restriction plays a more important role in controlling hepatic encephalopathy **(Grant *et al.*, 2021).** In this case report, hepatic formula diet (liver care), lactulose, metronidazole, S-adenosyl methionine and vitamin E; were used for 30 days. The puppy's diet was changed to a hepatic formula diet (liver care) with reduced protein to decrease the ammonia level in the blood. Lactulose was administered as an ammonia-reducing drug. Disaccharides such as lactulose reduce the absorption and increase the excretion of ammonia from the intestines. Lactulose is not absorbed from the intestine due to increased intestinal lumen acidity and reduced ammonia absorption **(Hudson and Schuchmann 2019).** In this study, Vitamins E and S-adenosyl-methionine have been used for their antioxidant and liver-protective properties.

Ultrasonography; has superiority over many diagnostic methods in terms of being non-invasive and not requiring anesthesia. In the last 20 years, the reliability and precision of ultrasonography have increased. Colour Doppler has replaced gray-scale ultrasound; this confirmed better identify the portosystemic shunt **(D'Anjou *et al.*, 2004).** Ultrasonography provides visualization of the hepatic parenchyma for examining portal vessels **(Cristopher, 1996).** The determination of PSS with ultrasonography has 47-95% sensitivity and 67-100% specificity, with accuracy reaching 94% in dogs and 100% in cats **(D'Anjou 2004).** In another study, **Lamb, (1996)** reported that the shunt was determined using ultrasound in dogs with portosystemic shunt and its sensitivity was 95% and specificity was 98%. Ultrasonography is one of the most definitive methods used to diagnose congenital portosystemic shunt and is sufficient to determine the presence of the shunted anomaly vessel **(Cristopher, 1996; D'Anjou *et al.*, 2004; Szatmari *et al.*, 2004).** In this case report, Portal flow velocity was 3.1 cm/s with pulsed-wave Doppler (PW-Doppler) with portal hypertension**. D'Anjou *et al.*, (2004)** reported that portal flow velocity was 17.8±4.4 cm/s in healthy dogs and three patients acquired PSS had a portal flow velocities of 2.9, 6.8, and 7.4 cm/s. The evidences of colour Doppler and portal flow velocity examination to diagnose the PSS was consistent with the findings of the authors mentioned above. The view of determining PSS with colour doppler was found to be consistent with the opinion of the authors mentioned above.

CONCLUSION

After the treatment, while ALT and BUN values were measured in the normal range, a significant decrease was determined in fasting serum bile acids and ammonia values. The clinical symptoms disappeared after the treatments. In addition to medical treatment, using chicken as a protein source in the hepatic formula diet (liver care) prevented the absorption of ammonia and helped to disappear the clinical symptoms. Portosystemic shunt is a very rare liver disorder in puppies. In this case report, as well as the importance of biochemical parameters in the diagnosis of a portosystemic shunt, importance of ultrasonographic imaging once again played an important role in determining the location of the shunt. In the diagnosis, the determination of turbulent flow with colour Doppler and decreased portal flow velocity with portal hypertension with PW-Doppler ultrasonography are the important diagnosis of the portosystemic shunt. It was concluded that without surgical operation, treatment resulted in disappear of clinical symptoms, use of lactulose, s-adenosyl methionine and a hepatic formula diet (liver care) may be beneficial.

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