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# Portography in dogs

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Summary

**The aim of the study was to assess the usefulness of vascular angiography in the diagnostics of liver vascular system diseases. The study was conducted on 3 dogs: Yorkshire terrier marked C1, German Shepherd – C2 and Airedale Terrier – C3, aged 11 months, 9 and 5 years respectively. The examinations were performed as follows: the history and clinical examination, abdominal USG, morphological examination, urea and creatinine concentration, AlAT, AspAT, ALP, GGT, amylase and lipase, the concentration of total bilirubin, ammonia, total protein and albumins. All animals underwent the angiography of the liver vascular system. Additionally, in laparotomy, an oligobiopsy of the liver was performed during which liver samples were collected for histopathological examinations. Results: Dogs C1 and C3 manifested leucocytosis. Only in dog C2 the morphological examination revealed thrombocytopenia. The biochemical examinations of blood serum in dog C1 showed a decrease in the urea level. Dog C2 demonstrated an increase the activity of AspAT, AlAT, ALP and GGT, as well as hyperbilirubinemia, hypoproteinemia and hypoalbuminemia. Dogs C1 and C2 had hyperammonemia. The histopathological examination of liver samples collected during diagnostic laparotomy in dog C1 revealed a slight fibrosis of single portal spaces and dilation of central veins and sinuses, which suggested passive hyperemia. Additionally, diffuse micro- and macrofollicular lipidosis of the whole bioptate was recognized. The histopathological examination of the collected liver bioptate in dog C2 showed macro- and micronodular cirrhosis of the liver. In dog C3 a venous congestion of the liver without signs of inflammation, fibrosis or lipidosis was diagnosed. The contrast examination of the liver vascular system in dog C1 revealed an extrahepatic portosystemic shunt. A connection of the splenic vein with the caudal vena cava in the form of a short loop between the portal vein branching off and the jejunal vein was observed. Dog C2 had multiple intrahepatic portosystemic shunts. In addition, a characteristic spiral course of intrahepatic branches was observed, which suggested liver cirrhosis. The examination of the liver vascular system in dog C3 revealed no abnormalities in the structure of the liver vascular system. Clinical signs and results of laboratory tests suggest the disease but the basic examination enabling the final diagnosis and location of a shunt is portography. This method is widely used in the diagnostics of liver vascular system diseases. However, it is an invasive method and should be performed in large specialist centres.**

**Keywords: dog, portography, liver**

Diseases of the liver vascular system constitute a common clinical problem in veterinary practice. One of the most frequently described diseases of the liver vascular system is a portosystemic shunt (PPS) (2, 4, 6-8). A portosystemic shunt in dogs was for the first time described by Hickam in 1949 (3). It may also occur in cats, foals, calves, miniature pigs and humans (3). The diagnostics of liver vascular system diseases is conditioned by a veterinary doctor's knowledge of anatomy. Blood reaches the liver via two main vessels: the hepatic portal vein (delivers about 75% of blood) and the hepatic artery (25% of blood) (9). The portal vein originates from the connection of the splenic vein and common mesenteric vein. It supplies the liver with blood rich in substances absorbed in the oesophagus lumen, among others: waste products, toxins (ammonia, phenols, indols, biogenic amines, etc.), enterohormones and other compounds originating in the oesophagus lumen under the influence of bacterial flora (5, 9). It is called a public vessel (*vas publica*) since the blood it supplies is used by the whole organism. The hepatic artery is a branch of the visceral artery branching off the aorta. It is called a private vessel (*vas privata*) since it supplies the liver with nutritious blood.

A portosystemic shunt is most often diagnosed in Miniature Schnauzers, Yorkshire Terriers and Irish Wolfhounds (6). In terms of their location, portosystemic shunts are divided into intrahepatic and extrahepatic. Extrahepatic portosystemic shunts account for 60% to 94% of clini-

cal cases and are most frequently diagnosed in small breed dogs. Intrahepatic portosystemic shunts account for 6-40% of all cases and are commonly diagnosed in large breed dogs (3). The basic examination of a portosystemic shunt location and type is a contrast examination of the liver vascular system: angiography, portography (9). The angiography is a modern technique of imaging of the liver vascular system, which enables detection of obstructions in the flow through these vessels as well as detection of congenital and acquired anomalies in their structure. The aim of the study was to evaluate the usefulness of vascular angiography in the diagnostics of liver vascular system diseases.

## Material and methods

The study was conducted on three dogs: Yorkshire terrier marked C1, German Sheperd – C2 and Airdale Terrier – C3, aged: 11 months, 9 and 5 years, respectively. The examinations were performed as follows: the history and clinical examination, abdominal USG, morphological examination (erythrocyte count, hematocrit value, haemoglobin concentration, leucocyte count with leucogram, thrombocyte count and erythrocyte indices (urea and creatinine concentration, activity of alanine aminotransferase (ALP), asparagine aminotransferase (AST), alkaline phosphatase (AP), y-glutamylotransferase (GGT), amylase and lipase, concentration of total bilirubin, ammonia, total protein and albumins). Next, the animals underwent the angiography of the liver vascular system. Prior to the examination the animals were subjected to 24-hour fasting. The dogs were premedicated with xylasine – 1 mg/kg b.w. and nalbufine – 0.1 mg/kg. Both preparations mixed in one syringe were given intramuscularly. After 10-15 min. a catheter was inserted into vena cephalica antebrachii. The animals were induced with propofol in dose 2-4 mg/kg b.w., and next intubated. Anaesthesia was maintained by a continuous infusion of propofol (0.4 mg/kg/min.). During the entire procedure passive oxygenotherapy and monitoring of basic life functions were performed by means of pulsoxymetry and capnometry. A catheter 18-20 G was inserted into the mesentenic vein to inject the contrast material (urografin 76%) in dose 1-2 ml/kg b.w. Next, the flow of blood in the liver vascular system was recorded with the aid of a fluoroscope for intraoperative X-ray examinations. Additionally, during laparotomy an oligobiopsy of the liver was performed to collect liver samples for histopathological examinations. The obtained preparations were stained with hematoxylin-eosin (HE), Giemsa method, according to Mallory and for the presence of reticulin fibres.

## Results and discussion

The clinical history revealed seizures in all the examined dogs. In one dog marked C1 the neurological signs intensified after eating. Blood carried to the liver by the portal vein contains many substances that in normal physiological conditions are metabolized in the liver. In animals with a portosystemic shunt these substances are not metabolized in the liver. Instead, they penetrate into the systemic circulation and cause toxic damage to the central nervous system (3, 6, 8, 9). The concentration of ammonia, mercaptans, B-hydroxylic biogenic amines, substances of benzodiazepine activity and other metabolites (GABA) rises. An increase in the concentration of these substances in CNU causes an impairment of synaptic stimuli transmission, the damage of neurons membrane and impairment of metabolism in the nervous tissue which leads to the emergence of encephalopathy symptoms (6). The clinical signs accompanying a portosystemic shunt are conditioned by an increase in intoxication of the organism: apart from seizures other non-specific neurological disorders may occur in animals, i.e. no response to external stimuli, purposeless walking, walking in circles, hallucinations, motor ataxia, dementia, metabolic tremor (characterized by occasional contractions of some muscle groups), and in more severe cases – coma (3, 9).

The animal with a portosystemic shunt may also experience gastroenterological disorders caused by an impairment of the liver synthesizing function (7). The clinical examination of the dog marked C2 revealed an enlargement of the abdomen outline. Additionally, on palpation, an increased tension of the abdominal muscles was noted and shifting dullness was heard on percussion. Ultrasound examination of dog C1's abdominal cavity revealed a decreased liver of homogeneous hyperechogenic structure with no focal changes and both kidneys enlarged. Ultrasound examination of dog C2's abdominal cavity showed fluid in the peritoneal cavity as well as a significantly decreased liver of heterogeneous echostructure and irregular margins. The dog marked C3 did not manifest any changes of the abdominal cavity organs in ultra-sound examination. Laboratory blood examinations in animals with a portosystemic shunt show in some cases microcyte anemia, which was not observed in the performed study (tab. 1). Dogs C1 and C2 manifested leucocytosis. Only in dog C2 morpho-

**Tab. 1. Test results of chosen morphological and biochemical parameters in blood of dogs C1, C2, C3**

| Parametr | C1 | C2 | C3 |
|---|---|---|---|
| Erythrocyte (t/l) | 7.01 | 6.79 | 7.74 |
| Hematocrit (l/l) | 0.49 | 0.42 | 0.53 |
| Haemoglobin (mmol/l) | 9.9 | 8.8 | 10.7 |
| MCV (f/l) | 70 | 62 | 69 |
| MCH (f/mol) | 1.51 | 1.3 | 1.39 |
| MCHC (mmol/l) | 21.2 | 20.9 | 20.5 |
| Leucocyte (g/l) | 18.4 | 13.9 | 22.2 |
| Limfocyte (%) | 11.2 | 4.6 | 15.0 |
| Monocyte (%) | 4.6 | 2.1 | 11.1 |
| Granulocyte (%) | 84.2 | 93.3 | 73.9 |
| Thrombocyte (g/l) | 459 | 133 | 320 |
| Urea (mmol/l) | 2.6 | 4.5 | 12.2 |
| Creatinine (µmol/l) | 119 | 83 | 124 |
| AspAT (U/l) | 33 | 131 | 23 |
| AlAT (U/l) | 26.0 | 129.5 | 62.0 |
| ALP (U/l) | 21 | 117.5 | 22 |
| GGT (U/l) | 10 | 35 | 10 |
| Ammonia (µmol/l) | 101 | 43 | 1 |
| Total bilirubin (µmol/l) | 1.7 | 32.0 | 1.7 |
| Total protein (g/l) | 68 | 34 | 65 |
| Albumin (g/l) | 35 | 18 | 34 |
| Amylase (U/l) | 1131 | 598 | 649 |
| Lipase (U/l) | 698 | 251 | 680 |

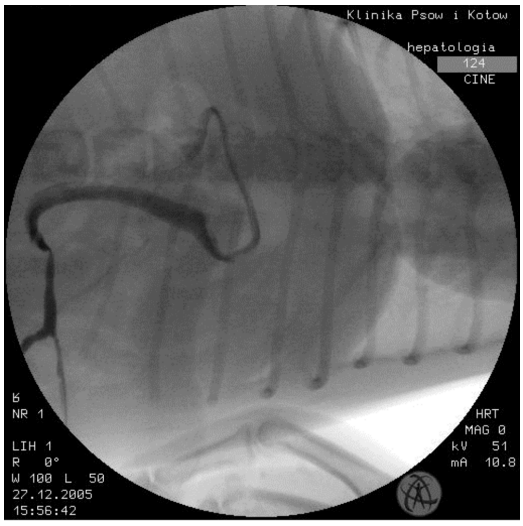


**Fig. 1. Extrahepatic portosystemic shunt in Yorkshire Terrier (C1)**

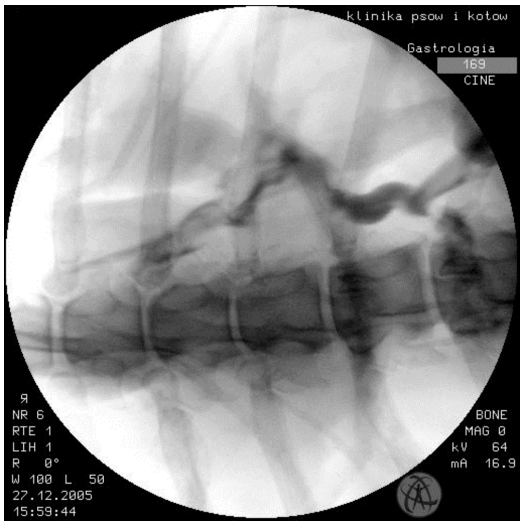


**Fig. 2. Multiple portosystemic shunts in dog with liver cirrhosis (C3)**

logical examination revealed thrombocytopenia, which is a typical sign of coagulation disorders in the course of liver diseases. The biochemical examinations of blood serum in dog C1 showed a decrease in the urea level. Dog C2 demonstrated an increase in AspAT, A1AT, ALP and GGT activity (tab. 1). In dogs with a portosystemic shunt a slight increase in alanine and asparagine aminotransferase is noted in some cases, yet the observed increase in hepatic enzymes in dog C2 suggested a serious liver damage (5). In addition, dog C2 demonstrated an increased concentration of total bilirubin and a decreased concentration of total protein and albumins, which is a specific sign of liver and bile duct diseases. Dogs C1 and C2 had hyperammonemia. Ammonia is a basic proteine catabolite in the intestines. The main location where ammonia originates as a result of proteolytic enzymes and bacterial ureases is the large intestine (2, 4, 7). In normal conditions ammonia originating in the intestines is metabolized in the liver into urea in the so-called Kreb's cycle. An increase in the concentration of ammonia in blood serum occurs in the course of chronic liver parenchyma diseases and also in cases of anomalies in the liver vascular system. The histopathological examination of liver samples collected in diagnostic laparotomy in dog C1 revealed slight fibrosis of single portal spaces and a dilation of central veins and sinuses, which suggested passive hyperemia. Additionally, diffuse micro- and macrofollicular lipidosis of the whole bioptate (20-25% hepatocytes) was recognized. The histopathological examination of the collected liver bioptate in dog C2 showed macro- and micronodular cirrhosis of the liver (*Cirrhosis macro- et micronodularis hepatis*).

In dog C3 fibrosis, lipidosis and a venous congestion of the liver without signs of inflammation were diagnosed. The contrast examination of the liver vascular system in dog C1 revealed an extrahepatic portosystemic shunt (fig. 1). A connection of the splenic vein with the caudal vena cava in the form of a short loop between the portal vein branch and the jejunal vein was observed. Dog C2 had multiple intrahepatic portosystemic shunts (fig. 2). In addition, a characteristic spiral course of intrahepatic branches was observed in this dog, which suggested liver cirrhosis. In the examination of the liver vascular system in dog C3 no abnormalities were found in the structure of the liver vascular system. In intrauterine life there is a connection between the portal vein and jejunal vein through the umbilical vein. This connection closes spontaneously within 3 days after birth under the influence of trombosan A2 released by the epithelium of blood vessels and the liver. If it fails to close, a permanent connection between the portal vein and systemic veins is formed and a portosystemic shunt is originated.

A portosystemic shunt is found primarily in large breed dogs. An acquired portosystemic shunt results from chronic liver diseases leading to portal hypertension. Numerous diseases that may initiate portal hypertension include, among others, chronic liver inflammation, liver cirrhosis, an arterio-venous shunt, portal vein thrombosis. Haemodynamic disorders in the portal system are usually secondary to the basic disease, yet in some cases they may occur spontaneously e.g. idiopathic portal hypertension in puppies. An increase in pressure in the portal system leads to its spontaneous relief through opening normally inactive connections between the portal system and the venous system of the systemic circulation. The portosystemic connections are subject to mutual hemodynamic relationships and constitute a natural way of collateral circulation regulating haemodynamic disorders in the liver vascular system. The number of originating connections depends on the duration of the primary disease and the location of the obstruction causing portal hypertension. Clinical signs and results of laboratory tests may suggest the disease but the basic examination that enables the final diagnosis and determination of shunt location is portography. This method is widely used in the diagnostics of liver vascular system diseases. However, it is an invasive method and should be performed in large specialist centres.

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