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Congenital portosystemic shunts in dogs

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Congenital portosystemic shunt is one of the commonest congenital abnormalities in dogs. The objective of our study was to determine the presence of portosystemic shunts in the dogs using computed tomography, point out the typical symptoms and changes in the blood parameters that occur during this pathology. We analyzed nine patients with portosystemic shunts and nine clinically healthy dogs. Each group contained four Yorkshire Terriers, two Miniature Poodles, two Maltese dogs, and one mixed-breed dog. The animals were clinically examined, underwent computed tomography, and their blood was analyzed. Computed tomography revealed congenital portosystemic shunts in the dogs of small breeds aged six months to four years. Of the seven patients that underwent computed tomography, five were diagnosed extrahepatic portosystemic shunts (two Miniature Poodles, one mixed-breed dog, one Maltese dog, and one Yorkshire Terrier) and two were found to have intrahepatic portosystemic shunt (Yorkshire Terrier and Maltese dog). According to the results of computed tomography, we determined that the connection of the shunting vessel occurred through the gastric, splenic, azygos, and phrenic veins with the caudal vena cava. In the plasma of the patients with portosystemic shunts, the ammonia concentration was five times higher. Hyperammonemia was closely associated with the complexity of the portosystemic shunt and the severity of pathological process. The ill dogs were observed to have typical symptoms of damaged central nervous system – apathy, stupor, sopor, muscle tremor, hyperkinesias, and static and dynamic ataxia. The nervous symptoms and increase in the ammonia content in blood are the symptoms that warrant computed tomography to verify the diagnosis of portosystemic shunts. The patients had impaired functions and the structure of the liver, especially notable in the parameters in blood serum, such as increased contents of total bile acids and bilirubin, reduced albumin content, and high activities of ALT, AST, and AP. Blood of a part of the ill dogs contained reduced contents of hemoglobin, number of erythrocytes, and hematocrit, increased contents of leukocytes and young forms of neutrophils, and also reduced number of platelets. In the future, we are going to analyze the efficacy of surgical treatment of dogs with portosystemic shunts.

Keywords: dogs; portosystemic shunt; ammonia; liver lesion; computed tomography.

Introduction

Portosystemic shunts occur in people (Kabaria et al., 2021; Umek et al., 2023), dogs (Moon et al., 2012; Phillips et al., 2025), cats (Sugimoto et al., 2018), horses (Willems et al., 2019), and cattle (Pietersma, 2018). Portosystemic shunts are formations of abnormal vessels in the portal venous system (White & Parry, 2015). Portal venous system is a network of vessels by which blood travels from the gastrointestinal tract to the liver. Nutrients, and also metabolites that form during their breakdown in the stomach and intestines, are metabolized and detoxified by the liver cells. Then, they return via hepatic veins to the circulatory system. As a result of the formation of anatomical abnormalities and pathological vascular connections between the portal venous system and the general venous blood flow, part of portal blood does not reach the liver (Caporali et al., 2015; Bahadori et al., 2022). Thus, metabolism products from the gastrointestinal tract, in particular endotoxins, are not neutralized by the liver cells and cause a toxic impact on the organism, infiltrating various organs and systems, and inducing pathological changes (Ronan al., 2020; Van den Bossche & van Steenbeek, 2020), in particular affecting the central nervous system (Gow, 2017; Jaffe et al., 2020).

Portosystemic shunts can be congenital or acquired, multiple or single depending on the number of pathological vessels, and microscopic or macroscopic depending on their sizes (Kraun et al., 2014; Konstantinidis et al., 2023b). Most often, this pathology is congenital, due to the limited detoxification function of the liver in fetuses in the perinatal period (Mankin, 2015; Nardelli et al., 2023). Thus, through a large shunt vessel, or a ductus venosus, blood bypasses the liver and enters the mother's circulatory system (Kim et al., 2021). After birth, this vessel closes, providing physiological hepatic blood circulation. If, for certain reasons, the ductus venosus remains open, the interhepatic portosystemic shunting persists (Cullen et al., 2021), and in case of formation of abnormal vessels between the cardiac and biliary veins of the fetus, an extrahepatic portosystemic shunt emerges (Nelson, 2011; Konstantinidis et al., 2023a).

Acquired portosystemic shunts form during the development of abnormal vessels in adult animals due to severe liver conditions, in particular cirrhosis, which leads to heightened pressure in the portal system (Butterworth, 2019; Rose et al., 2020).

Congenital and acquired portosystemic shunts need to be differentiated from other pathologies of different etiologies. Therefore, diagnostics of portosystemic shunts in animals involves a complex approach, taking into account clinical signs, laboratory blood parameters, and visual methods of study (Poggi et al., 2022). Clinical-laboratory diagnostics allow evaluating the general condition of ill dogs, and also determining the degree of organism intoxication (Vallarino et al., 2020; Kashliak & Vlizlo, 2023). Measuring the contents of ammonia, bile acids, and albumin, and also the activities of transaminases in blood of the patients is important for assessing the functional condition of hepatic cells (Vlizlo et al., 2023; Kozak et al., 2025).

Visual diagnostics allow determining the presence of portosystemic shunt. In particular, studies underscore the informativeness of ultrasound diagnostics, magnetic resonance imaging, and computed tomography (Ishigaki et al., 2016; Balda et al., 2025). Currently, computed tomography (CT) is considered a golden standard of quality visualization of portosystemic shunts (Farhoodimoghadam et al., 2024), particularly in dogs (Kurihara et al., 2024). The advantages of this method of diagnostics are its low invasiveness and high accuracy. Computed tomography allows localizing the abnormality and determining the anatomy of the pathological vessel (Parry & White, 2017; MacEwan & Thompson, 2023). An important aspect is the site of connection of the shunting vessel and its pathway to the caudal vena cava (Korol, 2022).

Computed tomography on small breeds of dogs is challenging due to reduced detailing because of small sizes of the examined animals. This is especially relevant for diagnosing congenital portosyste-

mic shunts, because CT is often performed on puppies, which complicates the identification of the site and pathway of the abnormal vessel even more. Moreover, veterinary doctors do not always have the opportunity to conduct CT scans on canine patients. The objectives of our study were to use computed tomography to determine the presence of congenital portosystemic shunts in dogs, demonstrate the significance of the ammonia level and typical symptoms of damaged central nervous system for diagnostics of the pathology and assessment of its severity, and also analyze the effects of the malformation on other organs and systems of the organism, particularly liver.

Materials and methods

All the procedures with the animals were conducted according to the European Convention for the Protection of Vertebrate Animals used for Experimental and other Scientific Purposes (Strasbourg, 1986) and the General Ethic Principles of Experiments on Animals, adopted by the First National Congress of Bioethics (Kyiv, 2001). The experiments were performed with adherence to the principles of humanity, outlined in the Directive of the European Community (Directive 2010/63/EU, 2010).

Dog owners brought their pets to the Merlion Clinic in Lviv for health issues such as the development of digestion problems (vomiting, diarrhea), apathy, enlarged abdomen, and neurological symptoms. At the clinic, the patients underwent clinical examination, and their blood was collected for laboratory studies, and computed tomography was performed. The blood samples were collected from the lateral subcutaneous vein of the forearm using a 21G needle, measuring 40 mm in length and 0.8 mm in width (Alexpharm, China). To obtain whole blood and plasma, the samples were collected in test tubes with heparine (Vacusel test tube with green cap, Turkey) and ethylenediaminetetraacetate (EDTA) (Vacusel test tube with violet cap, Turkey). The blood serum was obtained after collecting blood in Vacusel test tubes with yellow caps (Turkey), with a coagulation activator and gel. Blood (plasma and serum) were analyzed on the day when the animals were admitted. The whole blood was studied using a BC-30 Vet hematological analyzer manufactured by the company Mindray (Japan, 2020). In blood, we measured the concentrations of erythrocytes, hemoglobin, hematocrit, leukocytes, and platelets. The differentiation of leukocytes (leukogram) was performed after preparing smears and staining them according to Pappenheim and counting them in a special chamber under a microscope. The biochemical assays of the blood serum were carried out on a Mindray BS-240 automatic analyzer (Japan, 2020). In the blood serum, we determined the contents of total bilirubin, total bile acids (prior to feeding and two hours after), total protein, albumin, and urea, and also the activities of alanine aminotransferase (ALT), aspartate aminotransferase (AST), and alkaline phosphatase (AP). The ammonia concentration in the blood plasma was determined with a Fuji Dri-Chem NX500 biochemical analyzer (Japan, 2020).

To conduct computed tomography (CT), we used a Philips Brilliance 16 device (the Netherlands, 2005). Prior to performing CT, the dogs were sedated. For sedation, we used Dexdomitor 2–5 mg/kg (Orion Pharma, Finland), Butomidor 1–2 mg/kg (Gedeon Richter Ltd, Hungary), and Propofol 3–9 mg/kg (Novafarm-Biosyntez, Ukraine). During the examination, the dogs were in the lying position. We performed lateral and dorsal scanning from the cranial margin of the diaphragm to the hip joints. During CT, we paid special attention to the visualization of the liver parenchyma and hepatic vessels. If anatomic abnormalities were detected, individual regions of the liver were studied in more detail. The number of shunting vessels, as well as their beginning and ending points, was determined by visual assessment of the portal or caudal phases on different images.

In this study, we analyzed nine patients with portosystemic shunts, of which seven received CT scans. The number and breeds of the dogs were as follows: four Yorkshire Terriers, two Miniature Poodles, two Maltese dogs, and one mixed-breed dog. The clinically healthy dogs were selected so as to match the patients in terms of age and breed. The group of the clinically healthy dogs was formed of animals that were undergoing scheduled ambulatory examinations. The ill and clinically healthy dogs were of the following age: the Yorkshire Terriers were aged six months to three years, the Miniature Poodles were of the age of six months to three years, the Maltese dogs were aged one and three years, and the mixed-breed dog was of one year of age.

The blood parameters were statistically analyzed using Statistica 7 (StatSoft Inc., USA). The graphs were developed in Statistica 7 using the generally accepted algorithms. The article presents the mean arithmetic values and standard deviation x ± SD (mean ± standard deviation), as shown in in the figures. To compare the differences between the mean parameters of the clinically healthy and ill animals, we used the Tukey's Test, where the differences were considered statistically significant at $P < 0.05$.

Results

Upon admission to the clinic, the dog owners mentioned one or several reasons for the visit. In particular, the dogs had reduced activity, refused to eat fodder, vomited, suffered diarrhea, had enlarged abdomen, unsteady and uncoordinated gait, and cramps. During examination at the clinic, the patients exhibited various neurological conditions (apathy, stupor, and sopor). The dog patients suffered cramps, mostly as muscle tremors and hyperkinesia, and also impaired movement coordination, characterized by static and dynamic ataxia. At the same time, pain and tactile sensitivities persisted in all the studied animals.

The analysis of hematological parameters revealed that blood of the dogs with portosystemic shunts, compared with the clinically healthy dogs, had 13.8% lower hemoglobin content, 9.4% lower number of erythrocytes, and 11.1% lower hematocrit (Table 1).

When determining the number of leukocytes in blood of the ill dogs, we determined their 79.1% increase, compared with the clinically healthy dogs (Table 1). We should note that the number of leukocytes in blood of the shunt patients significantly fluctuated (4.5–44.4 10^9/L) and was high in half of the dogs, while in other half it was within or below the range of physiological fluctuations. The development of leukocytosis occurred due to a 3.24 increase in the relative number of band neutrophils.

Table 1
General analysis of blood of the dogs (x ± SE, n = 9)

Groups of animals	Hemoglobin, g/L	Erythrocyte, 10^{12}/L	Hematocrit, L/L	Leukocytes, 10^9/L	Eosinophiles, %	Band neutrophils, %	Segmented neutrophils, %	Lymphocytes, %	Monocytes, %	10^9/L Platelets, 10^9/L
Clinically healthy	170.9 ± 3.1	7.5 ± 0.2	0.45 ± 0.012	9.1 ± 0.6	2.4 ± 0.6	2.5 ± 0.6	69.9 ± 2.8	21.9 ± 2.5	3.2 ± 0.7	294.3 ± 26.4
Ill	147.4 ± 11.7	6.8 ± 0.5	0.40 ± 0.020	16.3 ± 4.1	1.8 ± 0.4	8.1 ± 3.3	66.0 ± 3.9	20.0 ± 3.8	4.1 ± 0.8	240.9 ± 38.4

Note: significant difference between the parameters in the table is absent.

The number of platelets in blood of the ill dogs decreased by 18.2% compared with the healthy dogs, and was low in six of the nine patients (Table 1).

The biological assays of blood of the dogs with portosystemic shunts revealed that the ammonia concentraton in plasma increased by 5.11 times ($P < 0.001$), accounting for 120.5 ± 20.1 μmol/L, as compared with 23.6 ± 3.4 μmol/L in the clinically healthy dogs. All the ill dogs had a high content of ammonia in blood, ranging 59.4 to 245.0 μmol/L (Fig. 1a). The content of total bilirubin in blood serum of the ill dogs (8.00 ± 1.03 μmol/L) was 40.8% higher (Fig. 1b), than in the clinically healthy dogs (5.68 ± 0.54 μmol/L) and in eight of the nine dogs exceeded the upper threshold of the reference values. Prior to feeding, the content of bile acids in blood serum of the clinically healthy dogs accounted for 11.13 ± 1.20 μmol/L, and after feeding it

was slightly higher, measuring 12.68 ± 1.64 μmol/L (Fig. 1c,d). In the ill dogs with portosystemic shunts, the concentration of bile acids in serum prior to feeding was 30.57 ± 12.20 μmol/L, i.e. 2.75 times higher compared with the clinically healthy dogs. After feeding, the content of bile acids in blood serum of the ill dogs (Fig. 1c, 1d) significantly increased (61.64 ± 12.31 μmol/L), being 2.02 times higher than prior to feeding, and also 4.86 times higher than in the clinically healthy dogs after a meal ($P < 0.001$). In blood serum of the ill dogs, the content of albumin decreased by 16.7% ($P < 0.01$), to 23.64 ± 1.55, compared with 28.36 ± 1.26 g/L in the clinically healthy dogs (Fig. 1e). In five of the nine ill dogs, the content of albumin decreased below the lower threshold of physiological fluctuations (25 g/L). This led to an insignificant decrease in the total protein in blood serum (from 71.2 ± 2.2 in the clinically healthy dogs to 64.8 ± 2.8 g/L in the ill dogs). The parameters of urea content in serum of the dogs with portosystemic shunts (5.88 ± 1.56 mmol/L) were no different from those of the clinically healthy dogs (5.82 ± 0.76 mmol/L).

The activity of amino transferase was significantly higher in blood serum of the dogs with portosystemic shunts. Thus, the ALT activity in blood serum of the shunt patients was almost 4.52 times higher ($P < 0.05$), measuring 233.5 ± 77.4 U/L, compared with 51.7 ± 4.5 U/L in the clinically healthy dogs (Fig. 2a). The activity of AST in serum of the ill dogs was 4.48 times higher ($P < 0.01$) than in the healthy dogs, equaling 175.3 ± 36.6 U/L, compared with 39.1 ± 4.6 U/L (Fig. 2b). The AP activity in blood serum was 216.7 ± 83.8 U/L in the ill dogs and 47.0 ± 6.3 in the clinically healthy dogs (Fig. 2c).

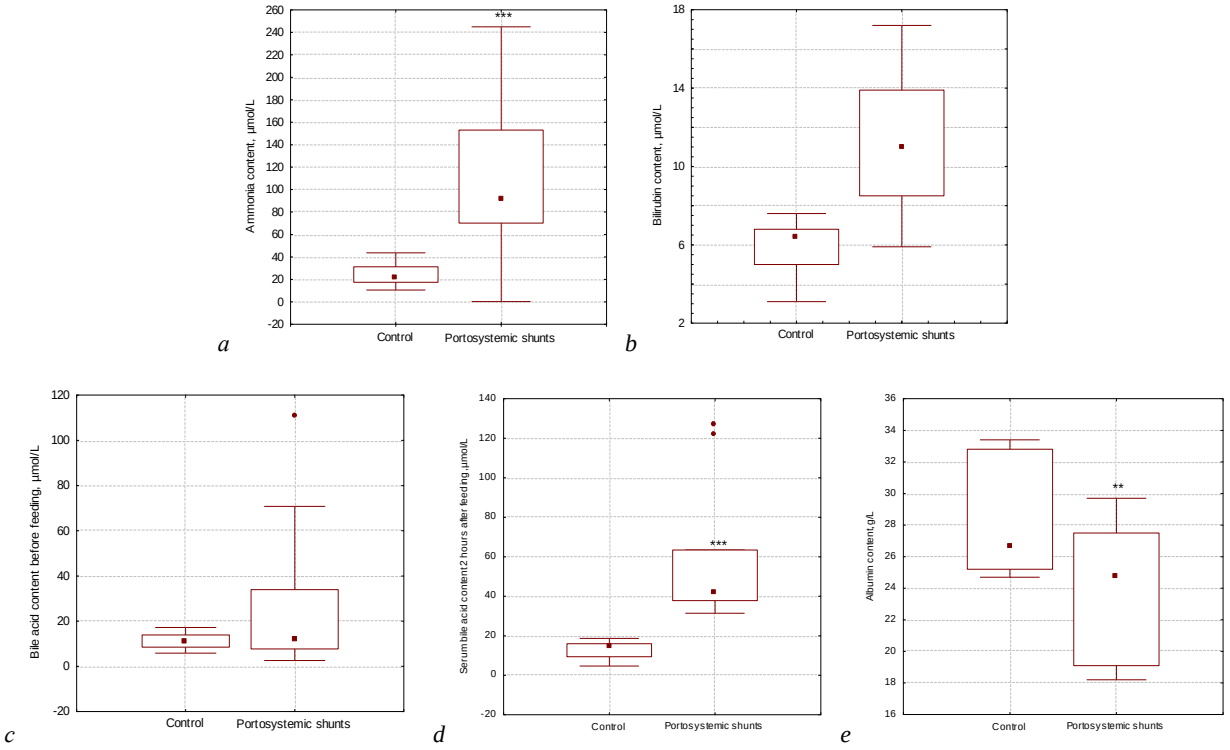

Fig. 1. Biochemical parameters of blood of the clinically healthy and ill dogs: *a* – ammonia content (μmol/L) in plasma; *b* – bilirubin content (μmol/L) in serum; *c* – concentration of bile acids (μmol/L) in serum prior to feeding; *d* – content of bile acids in serum two hours after feeding (μmol/L); *e* – albumin content (g/L) in serum; the abscissa axis indicates groups of animals and the ordinate axis indicates the units of parameter mesurements; small square – median, upper and lower rectangle borders – 25% and 75% quartiles, vertical line – minimum and maximum values, circles – outliers; n = 9; ** $P < 0.01$; *** $P < 0.001$

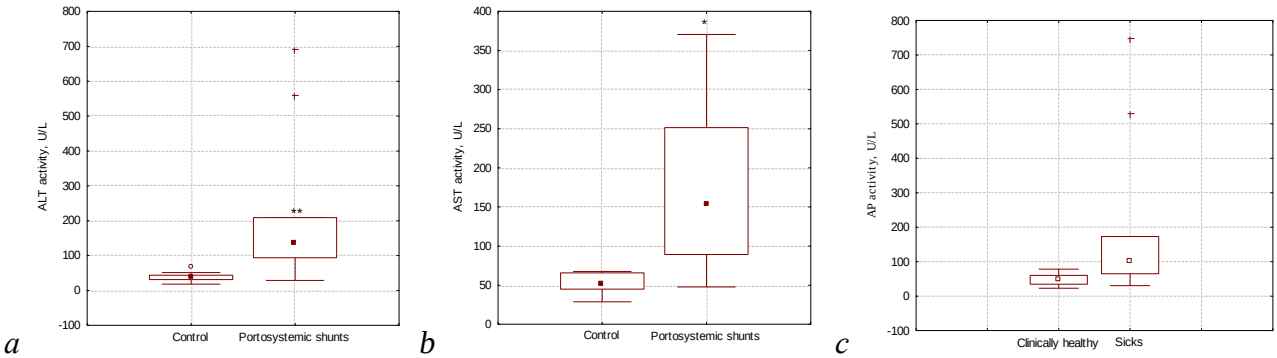

Fig. 2. Activity of enzymes in blood serum of the dogs (U/L): *a* – ALT, *b* – AST, *c* – alkaline phosphatase (AP); the abscissa axis indicates groups of animals and the ordinate axis indicates units of parameter measurements; small square – median, upper and lower rectangle borders – 25% and 75% quartiles, vertical line – minimum and maximum values, circles – outliers; n = 9; * $P < 0.05$; ** $P < 0.01$

Computed tomography of the dogs revealed extrahepatic portosystemic shunt in five of the seven patients and intrahepatic portosystemic shunt in two patients. Extrahepatic portosystemic shunt was found in two Miniature Poodles, the mixed-breed dog, one Maltese dog, and one Yorkshire Terrier. Intrahepatic portosystemic shunts were diagnosed in one Yorkshire Terrier and one Maltese dog.

If an extrahepatic portosystemic shunt was present, the shunting vessel had a dorsal course, and the connection of the gastric vein (vena gastrica) occurred after its exit from the portal vein, bypassing the liver (Fig. 3a). In the other cases, extrahepatic portosystemic shunt was characterized by the connection of gastroduodenal vein to the portal vein (vena portae). The extrahepatic abnormal vessel started its

path from the portal vein, then passed through the splenic vein (vena lienalis) and connected to the azygos vein (vena azygos). Ventrally from the aorta and medially from the stomach, as seen in the transverse CT section in the image, we observed an aberrant (atypical) vessel that gradually ramified from the portal vein but did not enter the liver (Fig. 3b). While examining the transverse CT section of the dogs with intrahepatic portosystemic shunts, there were seen shunting vessels passing through the left side of the liver, travelling toward the caudal vena cava (vena cava caudalis), and draining into it (Fig. 4a, 4b). For the other cases, the right side of the liver is pictured, in which shunt is bent tortuously, forming a loop, and thus a mutual connection in the form of a branch of the right portal vein (vena portae), draining into the right lateral hepatic vein (vena hepatica lateralis, Fig. 4c).

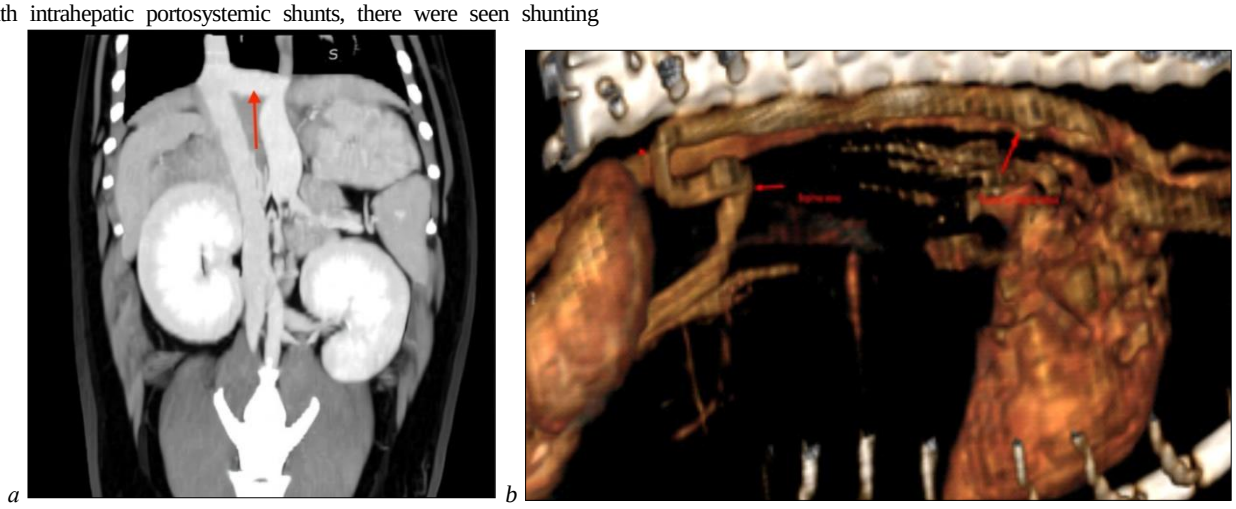

Fig. 3. Computed tomography of extrahepatic portosystemic shunt; red arrows indicate the shunting vessels: *a* – vena gastrica bypasses the liver; *b* – aberrant shunting vessel

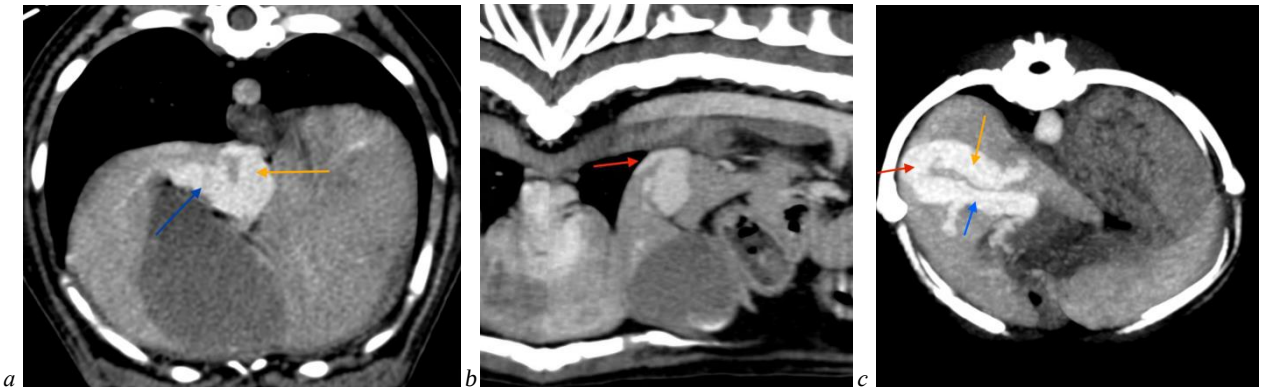

Fig. 4. Computed tomography of the intrahepatic portosystemic shunt: *a* – yellow arrow indicates vena cava caudalis, blue arrow – drainage from vena portae into vena cava caudalis; *b* – red arrow – shunting vessel; *c* – blue arrow – vena portae; yellow arrow – vena cava caudalis, red arrow – shunting vessel

Discussion

Congenital portosystemic shunts are diagnosed in small breeds of dogs, in particular Yorkshire Terriers, Miniature Schnauzers, Chihuahuas, Labrador Retrievers, Poodles, Pugs, Dachshunds, Cocker Spaniels, and Pomeranian Dogs (Lidbury et al., 2015). Of the dogs we examined, pathology was discovered in Yorkshire Terriers, Miniature Poodles, mixed-breed dogs, and Maltese dogs. Those are small breeds of dogs, and also young animals, and therefore we can assume that their portosystemic shunts were congenital. Congenital portosystemic shunt is one the commonest congenital abnormalities in dogs (Mankin, 2015; Seller et al., 2022). This pathology is characterized by the development of abnormal vessels that connect the portal vein or its tributaries with the systemic circulation. Those vessels allow the venous blood that enters from the gastrointestinal tract, pancreas, and spleen to bypass the liver and enter directly the systemic circulation (Berent & Tobias, 2018). As a result, various endotoxins such as ammonia, mercaptans, free fatty acids, phenols, and bile salts from the gastronintestinal tract are absorbed, are not completely metalobilized or removed by the liver, and thus reach the circulatory system and gradually accumulate in blood, causing intoxication (Spillane et al., 2021; Lidbury, 2025). Ammonia is the main endotoxin whose increase in blood of patients suggests a presence of portosystemic shunts (Tivers et al., 2014; Caporali et al., 2015). The studies we conducted demonstrate that the ammonia concentration in blood plasma of the dog patients with portosystemic shunts increased by over five times. Ammonia is most harmful to the central nervous system (Kawaguchi et al., 2019; European Association, 2022), where it impairs the function of astrocytes and causes the development of oxidative stress, leading to brain edema and intracranial hypertension (Williams et al., 2020). Hyperammonemia was closely associated with the complexity of portosystemic shunt and the severity of the pathological process in dogs.

Clinical signs of the pathology can be of limited diagnostical value, because the course of the pathology could be characterized by general symptoms and alternation of periods of gaining strength and weakening (Bedford, 2017; Farhoodimoghadam et al., 2024). Exacerbation of the pathology manifests in symptoms indicating affected central nervous system, gastrointestinal tract, and urinary tract (Konstantinidis et al., 2023a). Typically, only then do the pet owners seek help at the clinic. According to our data, and also reports of other researchers, dogs with portosystemic shunts are observed to have depression, weakness, ataxia, circular movements, head pressing, cortical blindness, cramps, and coma (Krishnarao & Gordon, 2020; Konstantinidis et al., 2023b). Moreover, patients suffering this pathology have enlarged liver and pains in it. Studies of the biochemical parameters of blood from the ill dogs revealed changes characteristic of liver pathology. Thus, blood of the dogs with portosystemic shunts,

besides significant increase in the ammonia concentration, contained high contents of bile acids and bilirubin. At the same time, the content of albumin was decreased, while the activities of ALT, AST, and AP were elevated. Such changes in blood were described in the article by Vallarino et al. (2020).

It has to be noted that the concentration of bile acids in blood serum of the dogs with portosystemic shunts increased, especially after feeding. Cholemia was notable in all the ill dogs. To a lesser degree, there was observed increase in the content of total bilirubin in blood serum. However, if taking into account the reference values of bilirubin in the serum, then eight of the nine patients had the parameters exceeding the determined upper threshold. These changes indicate that in dogs with portosystemic shunts, liver cells are affected, which therefore ineffectively transport bile acids and bilirubin from hepatocytes to bile pathways, causing increased influx of these metabolites in blood (Lester et al., 2016; Vlizlo et al., 2023). Also, it is also possible that significant increases in the concentrations of bile acids and bilirubin in blood of the ill animals can have a toxic effect on the central nervous system. Affected hepatocytes and impairment of the protein-synthesizing function of the liver were indicated by the decrease in the content of albumin in blood serum from the dogs with portosystemic shunts. Hypoalbuminemia has indeed been observed as a characteristic of dogs suffering from portosystemic shunts (Moritz, 2014).

Despite the significant increase in the ammonia concentration in blood plasma of the dogs, the content of urea in serum did not change. This is related to the fact that ammonia bypasses the liver, and as a result concentrates in blood, and therefore its increase is not related to the impaired neutralization in the liver and formation of urea. Moreover, the urea-forming function of hepatocytes is quite stable, but can be impaired during a significant damage to the liver parenchyma (Webster, 2017).

Damage to the liver cells in dogs suffering portosystemic shunts can be indicated by high activities of amino transferases (ALT, AST) in blood serum. Alanine and aspartate aminotransferases are not purely specific enzymes indicating liver damage, but their activities in blood are always high in animals with hepatic conditions (Simonov & Vlizlo, 2015; Lawrence & Steiner, 2017; Zelenina et al., 2022).

Simultaneous presence of clinical signs and blood parameters characteristic of affected liver and central nervous system suggests the development of hepatic encephalopathy (Kashliak & Vlizlo, 2024). At the same time, ammonia plays a key role in the pathogenesis of hepatic encephalopathy (Lima et al., 2019; Bellafante et al., 2024). Furthermore, disorders in the main liver functions, and also decreased influx of nutrients through the portal blood flow from the gastrointestinal tract to hepatocytes can cause their deficit in the organism and aggravate the pathology.

To corroborate the diagnosis of the presence of portosystemic shunt in dogs, the patients must be referred to computed tomography (Takeuchi et al., 2025). Therefore, dogs admitted to the clinic with cramps, depression, ataxia, pain in the liver region, and exceptionally high parameters of ammonia in blood must undergo CT. As indicated by CT results, the connection of shunting vessel can develop through gastric, splenic, azygos, and phrenic veins, and the caudal vena cava. The commonest anatomic types of portosystemic shunts in dogs are splenic-phrenic shunt, splenic-azygos, right gastric-caval, splenic-caval, right gastric-caval with a caudal loop, right gastric-phrenic, colon-caval, splenic-phrenic, portal-caval, right gastric, and portal-caval (Fukushima et al., 2014; White et al., 2018). Also, a pathological communication can occur starting from the right gastric vein and connect with the left gastric vein, travelling through the lesser curvature of stomach. Gastric azygos shunt is characterized by the start from the left gastric vein and connection with the right azygos vein. After exit of the shunt from the portal vein, often its decrease in diameter is registered.

Conclusions

Congenital portosystemic shunts were diagnosed in young (aged six months to four years) and small dog breeds (Yorkshire Terrier, Miniature Poodles, mixed-breed dog, Maltese dog). The presence of abnormal vessels connecting the portal vein with the circulatory system, bypassing the liver, leads to the accumulation of endotoxins in the organism. These, first of all, include ammonia, which penetrates the blood-brain barrier and causes the development of typical symptoms of damaged central nervous system – apathy, stupor, sopor, muscle tremors, hyperkinesias, and static and dynamic ataxia. In the ill dogs, other vitally important organs and systems are affected as well. In particular, impairments occur in the main functions and structure of the liver (cholemia, hyperbilirubinemia, hypoalbuminemia, and high activities of ALT, AST, and AP). The contents of hemoglobin, hematocrit, the number of erythrocytes decrease. Furthermore, leukocytosis, neutrophilia, and thrombocytopenia emerge. Ill dogs with high level of ammonia in blood and emerging symptoms of pathology of the central nervous system are referred to computed tomography imaging in order to confirm or disprove the diagnosis of the presence of intrahepatic or extrahepatic portosystemic shunt.

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