Acta Scientiae Veterinariae, 2023. 51(Suppl 1): 857.

CASE REPORT
Pub. 857

ISSN 1679-9216

Congenital Extrahepatic Portosystemic Deviation of Gastric Vein with Azygos in a Bitch

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ABSTRACT

***Background*:** Portosystemic shunt (PSS), an alteration commonly found in toy dogs, is caused by an anastomosis between the systemic and portal circulation, interfering with the metabolism of several toxins. It can be of congenital or acquired origin and is classified as intra- or extrahepatic. Clinical signs include the gastrointestinal tract, nervous system, and urinary system according to the fraction of the shunt. It is diagnosed by several imaging tests and exploratory laparotomy. Therapy involves drug therapy and/or surgical correction of the anomalous vessels. Thus, the aim is to present an unusual case of extrahepatic cPSS originating from the left gastric vein and insertion into the azygos vein.

***Case*:** A 2-year-old female toy poodle, spayed, weighing 2.7 kg was treated with a history of recurrent cystitis and neurological signs such as focal seizures, ataxia, tremors, blindness, lethargy, head pressing, and compulsive gait. Complementary tests revealed normochromic microcytic anemia, neutrophilia-induced leukocytosis, monocytosis, and lymphopenia. Biochemical analysis revealed hypoproteinemia due to hypoglobulinemia, an increase in alanine aminotransferase, aspartate aminotransferase, alkaline phosphatase, and gamma-glutamyl transferase, and a decrease in urea. In the urinalysis, ammonium biurate crystals were detected, and Doppler ultrasound revealed microhepathy and the presence of an anomalous gastrosplenic vein inserted into the azygos vein, a finding compatible with the congenital extrahepatic PSS. Abdominal tomography confirmed vascular deviation with a sinuous path originating from the left gastric and splenic veins, inserting into the azygos vein, measuring approximately 5.95 cm in length. Cranial tomography revealed changes consistent with hepatic encephalopathy. Drug therapy was performed with hydration, liver chow, lactulose, probiotics, metronidazole, S-adenosyl-L-methionine, and ursodeoxycholic acid, and after 15 days, surgery was performed to place a 3.5 mm ameroid constrictor ring for gradual occlusion of the anomalous vessel. The animal recovered well, and a control abdominal ultrasound was repeated 30 days after the procedure, noting that the constrictor had not yet fully occluded the deviation. Doppler imaging revealed a favorable evolution with an increase in the diameter of the portal vein in the hepatopetal direction. The patient was followed-up for a year and had a normal and healthy life.

***Discussion*:** Extrahepatic PSS is frequently diagnosed in purebred and toy dogs, commonly occurring between the portal vein and one of its tributaries, with a lower frequency of anomalous vessels between the azygos veins, as in the present report. The patient's age and clinical signs were compatible with the disease, in addition to ammonia biurate crystals and hematological and biochemical alterations. The neurological clinical signs observed were compatible with hepatic encephalopathy secondary to congenital PSS. The imaging examinations facilitated the identification of the extrahepatic vascular anomaly, with the tomography being more accurate and helping in proper surgical planning. Clinical treatment should be performed for presurgical stabilization, and occlusion can be performed by placing cellophane bands or an ameroid constrictor, which is the technique of choice for congenital PSS, as it allows for slow constriction to avoid acute portal hypertension, as in this case, emphasizing that anesthesia in animals with portosystemic shunts must be performed with care.

Keywords: dog, surgery, portosystemic shunt, ameroid ring, gastric vein, azygos vein.

DOI: 10.22456/1679-9216.127435

Received: 26 November 2022 Accepted: 1 March 2023 Published: 8 April 2023

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INTRODUCTION

A shunt or portosystemic shunt (PSS) is a vascular communication between the portal and systemic circulation that diverts the blood flow through the liver. It can be congenital or acquired [8]. Congenital PSS (cPSS) is caused by embryonic vessels, in which a shunt occurs without association with portal hypertension. Conversely, acquired portosystemic shunting is an anastomosis formed owing to portal hypertension [4].

The cPSS may be extra- or intra-hepatically present. Extrahepatic cPSS are anomalous vessels that connect the portal vein or one of its contributors to the caudal vena cava or azygos. Intrahepatic cPSS results from failure to close fetal communication or the persistence of ducts that communicate the umbilical vein to the vena cava [2,12].

The clinical signs are the most varied and are related to liver dysfunction in animals [9]. In animals with cPSS, signs are observed before the first year of life; however, some can manifest as clinical signs in adulthood [2].

PSS can be definitively diagnosed using abdominal Doppler ultrasound, computed tomography, contrast radiography, exploratory laparotomy, and portal transcolonic scintigraphy [1].

This report presents an unusual case of extrahepatic cPSS originating from the left gastric vein and insertion into the azygos vein, highlighting the clinical, surgical, laboratory, and imaging aspects.

CASE

A 2-year-old spayed toy poodle bitch weighing 2.7 kg was treated for emesis after feeding, ataxia, focal seizure, head pressing, lethargy, and circling. The patient was hospitalized because of seizures and a history of cystitis. A full blood count, urinalysis, and imaging tests were performed.

The blood count showed microcytic (51.7 fL) normochromic (38.7 g/dL) anemia (Ht 29.7%), leukocytosis (51,900/mm^3) due to neutrophilia (48267/mm^3) and monocytosis (3114/mm^3), and lymphopenia (519/mm^3). Biochemical examination revealed hypoproteinemia (5.26 g/dL) due to hypoglobulinemia (2.34 g/dL); increased alanine aminotransferase (141 IU/L), aspartate aminotransferase (92.3 IU/L), alkaline phosphatase (294.3 IU/L), and gamma-glutamyl transferase (7.23 IU/L); and decreased urea (20.4 mg/dL). On urinalysis, mild hematuria and ammonium biurate crystals were observed.

Ultrasound examination revealed that the liver had reduced dimensions, with homogeneous and diffuse hyperechogenic parenchyma. The edges were tapered and regular, suggesting a micropathy. Abdominal Doppler revealed an anomalous extrahepatic vessel originating from the gastrosplenic vein inserted into the azygos vein. A decrease in the portal vein caliber was observed in the portohepatic segments.

Computed tomography of the liver revealed cross-sections of 3 mm and increment of 1.5 mm, with multiplanar reconstructions, pre- and post-contrast; an anomalous vessel that originated at the bifurcation of the left gastric vein and splenic vein and inserted into the azygos vein. This vessel measured 4.21 mm in diameter at the point of insertion between the veins, was quite sinuous, left the mid-abdomen region, and was followed in a dorsal cranial direction until it was inserted into the azygos vein at the level of the 12th thoracic vertebra. At the point of insertion into the azygos vein, the anomalous vessel measured 3.09 mm in diameter and was approximately 5.95 cm long between its origin and insertion (Figure 1A, B, & C). The liver was reduced in size; however, had a regular shape and contour. Nonobstructive nephrolithiasis was observed in the left renal pelvis. Cranial tomography showed hypodensity in the right frontal lobe (Figure 2A) and bilateral thalamus, without the presence of a mass.

Based on these findings, extrahepatic cPSS was diagnosed with the origin in the left gastric vein and insertion in the azygos vein, and surgical treatment was instituted for the placement of the ameroid constrictor ring in the diversion vessel. Presurgical stabilization started 15 days before the procedure, and liver care diet[1] [Royal Canin®] was prescribed, lactulone [2] [lactulose® - 1 mL/4 kg BID for 5 days], probiotic[3] [Vetnil® - 1 g SID, for 2 days), Metronidazole[4] [Prati-Donaduzzi® - 15 mg/kg BID], SAME[5] [Nutri Same® - 20 mg/kg SID], and Ursacol[6] [Ursodeoxycholic Acid DrogaVET® - 10 mg/kg SID]. The animals showed a significant improvement after the beginning of clinical treatment.

In the anesthetic protocol, induction was performed with propofol[7] [Propovan® - 6 mg/kg IV dose/effect] and maintenance with remifentanil[8] [Remifentanil Hydrochloride® - 10 mcg/kg/h IV] in continuous infusion and isoflurane[9] [Isoforine®] as volatile anesthesia. For pain control, dipyrone[10] [Dipyrone Monohydrate® - 25 mg/kg IV], methadone[7] [Methadone

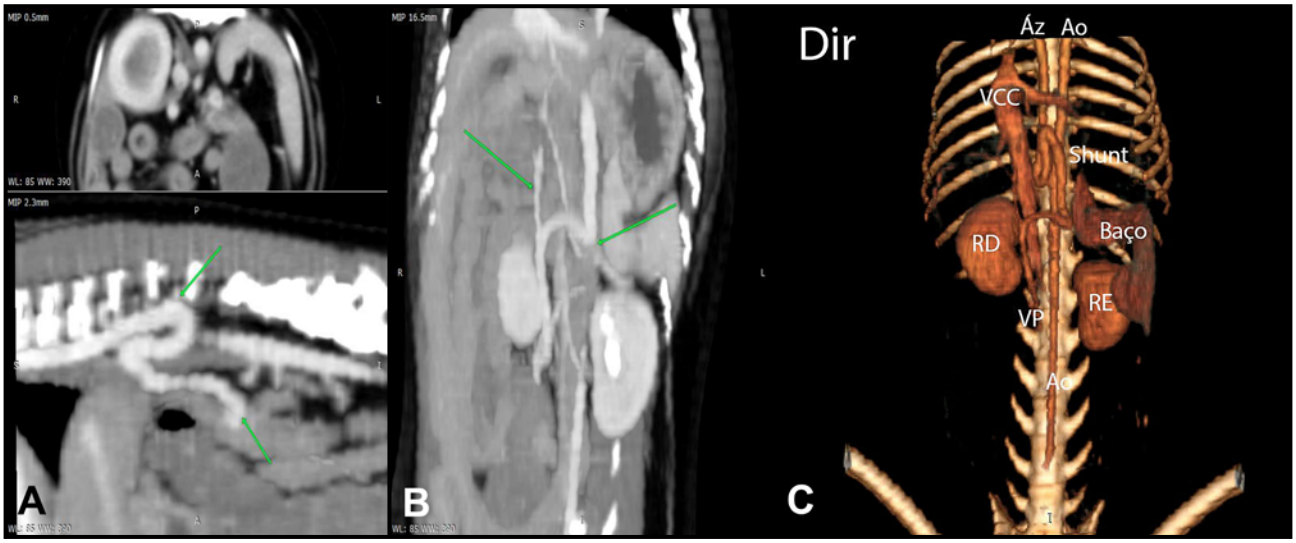

Figure 1. The congenital extrahepatic portosystemic shunt of the gastric vein with azygos in a 2-year-old spayed toy poodle bitch. A- B- & C- The post-contrast computed tomography of the liver in 3 mm and 1.5 mm pitch cross-sections and multiplanar reconstructions. In the post-contrast phase, an anomalous blood vessel of tortuous trajectory isobserved, originating from the bifurcation of the left gastric and splenic veins, which is inserted into the azygos vein. This vessel measures 4.21 mm in diameter at the point of insertion between the veins, is sinuous and left the mid-abdomen region, and followed in a dorsal cranial direction until it is inserted into the azygos vein, at the height of T12 (arrows). At the point of insertion into the azygos vein, the anomalous vessel measures 3.09 mm in diameter and is approximately 5.95 cm long between its origin and insertion.

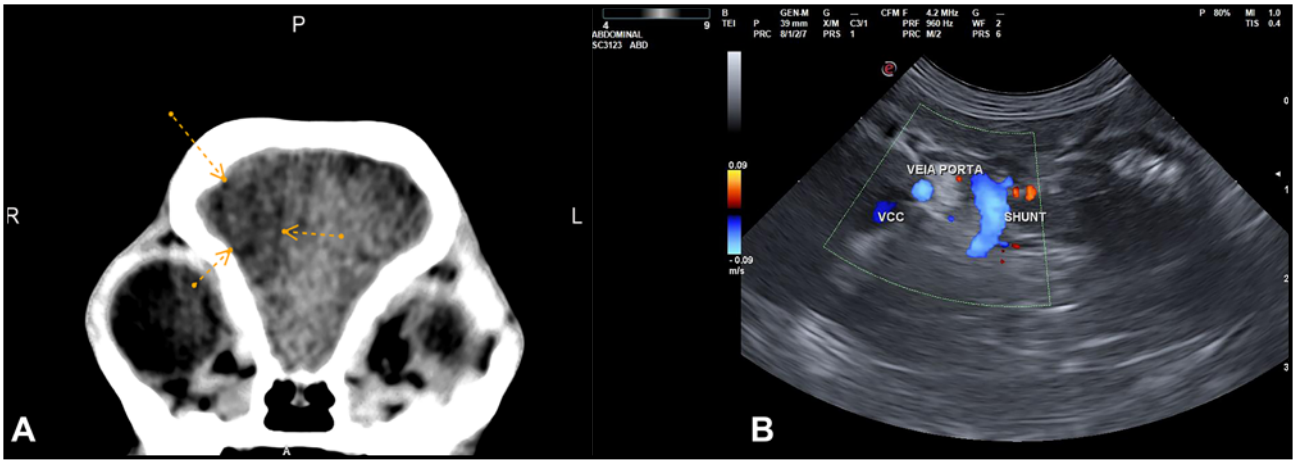

Figure 2. The congenital extrahepatic portosystemic shunt of the gastric vein with azygos in a 2-year-old spayed toy poodle bitch. A- Skull tomography shows hypodensity in the right frontal lobe (arrows). B- Transverse abdominal ultrasound 30 days after placement of the ameroid ring. There is still little hepatofugal flow with the azygos vein owing to partial occlusion of the annulus.

Hydrochloride® - 2 mg/kg IV], and maxicam[11] [Meloxicam® - 0.1 mg/kg IV] were administered.

Perioperatively, the animal was positioned in the dorsal decubitus position, and trichotomy and antisepsis were performed in the entire ventral region of the abdomen and thorax. An abdominal incision was made in the ventral midline, caudal to the xiphoid cartilage. Self-static retractors were used to facilitate exposure of the abdomen and visualization of the anomalous vessel that had its origin in the left gastric and splenic veins, with insertion at the beginning of the formation of the azygos vein. The bypass vessel was carefully dissected and anchored using 3-0 nylon[12], allowing placement of a 3.5 mm ameroid constrictor ring. Micropathies and alterations in the splenic parenchyma were observed.

One month after the placement of the ameroid ring, a control ultrasound revealed that the constrictor did not completely close the anomalous vessel. The liver continued with reduced dimensions, heterogeneous and diffuse hyperechoic parenchyma, and thin regular edges. Doppler showed a favorable evolution, with an increase in the diameter of the portal vein and hepatopetal flow; however, in the PSS, there was still evidence of moderate hepatofugal flow to the azygos vein (Figure 2B). One year postoperatively, the animal showed no further clinical changes.

DISCUSSION

In this study, we observed the occurrence of a rare extrahepatic cPSS originating in the left gastric and splenic veins with insertion into the azygos vein. In general, extrahepatic PSS connects the portal vein to the caudal vena cava (portocaval) or the portal vein to the azygos vein (portoazigos) [7]. The gastric vein PSS with azygos vein was observed to occur more rarely in animals in several retrospective studies or case series, in which only one animal presented the deviation [7].

Doppler ultrasound facilitated the evaluation of the size of the liver and the observation of extrahepatic deviation. Ultrasonography is a useful test for detecting PSS as it allows the evaluation of tissue vascularization [3]. Additionally, ultrasonography can assist in measuring anomalous vessels, which is important for the proper choice of the diameter of the ameroid ring to be used in surgical correction [14]. The microhepatia were visualized by radiographic examination. This hepatic alteration occurs because of the absence of hepatotrophic substances from the pancreas and intestine, which is a common alteration in animals with PSS [6].

Computed tomography is an excellent method for locating anomalous vessels in animals with PSS. This technique helps confirm the diagnosis, provides an accurate vascular map and identifies complications and associated malformations [14]. Tomography in this study is fundamental for choosing the best treatment method for patients.

The neurological clinical signs observed were compatible with hepatic encephalopathy secondary to cPSS. This syndrome is referred to as central nervous system dysfunction owing to liver failure [5]. The pathogenesis of hepatic encephalopathy is not well understood. Nevertheless, the most common suggestions include the role of neurotoxins, impaired neurotransmission due to metabolic changes in liver failure, changes in brain energy metabolism, systemic inflammatory response, and alterations in the blood-brain barrier [3]. Furthermore, the development of clinical signs in animals with PSS is associated with the amount of blood diverted from the portal to the systemic circulation [7]. The anomalous vessel in this case had a caliber of 4.21 and 3.09 mm in diameter at its point of origin and insertion, respectively. This vessel opening diameter probably favored a greater volume of blood diverted from the portal to the systemic circulation, causing the observed clinical signs.

The brain tomography of this case showed hypodensity in the right frontal lobe and bilateral thalamus, without the presence of a mass. These findings are compatible with a greater accumulation of fluid in the nervous tissue (cerebral edema), which may have favored the onset of clinical signs. Cerebral edema is a common neurological disorder in patients with hepatic encephalopathy due to a rapid increase in ammonia levels. At high levels, ammonia can cross the blood-brain barrier, where astrocytic glutamine synthetase converts ammonia and glutamate into glutamine, which in turn acts as an osmolyte and increases cerebral volume [13].

The hematological and biochemical changes observed in this study were similar to those described in other dogs with cPSS [7]. The presence of ammonium biurate crystals on urinalysis is common in dogs with PSSs. The presence of these crystals can be explained by the increase in ammonia and uric acid resulting from the cPSS [10]. These crystals can trigger clinical signs such as dysuria, pollakiuria, hematuria, and stranguria [2,14]. Additionally, they may predispose patients to lower urinary tract infections [7], as observed in this case.

Anesthesia in animals with PSS should be performed carefully because of liver dysfunction and hepatofugal flow that impair drug clearance [6].

In this case, surgical treatment consisted of placing an ameroid constrictor ring. This technique allows gradual occlusion of the shunt, as abrupt closure of the anomalous vessel can cause acute portal hypertension [3]. The cellophane band is also used to correct the PSS, as it causes the vessel to gradually close. Occlusion occurs due to an inflammatory reaction in the foreign body and perivascular fibrosis [11]. The animal in this study responded well to the surgical procedure and returned to normal feeding without the need for therapeutic feed and clinical manifestations 1 year after the surgery.

It is noteworthy that congenital shunts are more commonly observed in animals up to 1 year of age [3]. In this study, it was observed in a two-year-old poodle toy. Congenital PSS should be included in the differential diagnosis of older animals with neurological clinical signs.

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***Acknowledgements*.** This study was financed in part by the Coordenação de Aperfeiçoamento de Pessoal de Nível Superior - Brasil (CAPES) - Finance Code 001. This research was also financially supported by Fundação de Amparo à Pesquisa e Inovação do Espírito Santo - FAPES.

***Declaration of interest*.** The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

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