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A PORTOSYSTEMIC SHUNT IN A DOG: HAS ITS ANATOMY CHANGED?

A congenital portosystemic shunt (CPSS) is a vascular connection between the portal and the systemic venous circulation bypassing the liver. These connections are abnormalities formed during the embryological development of the liver and the abdominal venous system. The abdominal veins derive from three embryonic vascular segments: the umbilical, the vitelline and the cardinal veins. Usually a CPSS is present as a single vessel, but in some dogs two or more congenital shunts are seen.

In dogs congenital shunting is rare. Less than 0.2% of all dogs are reported to develop a CPSS, although the odds are higher in breeds like Yorkshire terriers, pugs, Maltese and miniature schnauzers. The large majority consists of extrahepatic shunts (EH), which are usually seen in small breeds, whereas the intrahepatic shunts (IH) are less frequently diagnosed and are mostly described in large breeds⁽¹⁾. In the Netherlands the most common breeds with an extrahepatic shunt are probably the Jack Russell terrier, the Maltese, the dachshund, the Yorkshire terrier and the Chihuahua; the most common breeds that are seen with an intrahepatic shunt are the golden retriever, the Labrador retriever and the Bernese mountain dog. Besides the extrahepatic and intrahepatic location of a CPSS, shunts can be divided into several subtypes. A correct anatomical diagnosis of a CPSS is important for a clinician to optimise (surgical) treatment and to establish a reliable estimation of the prognosis.

Extrahepatic shunts are traditionally described as porto-caval or porto-azygos shunt, depending on their outlet that is usually visualised using ultrasonography and confirmed during surgery^(1,2). In dogs, the proportion of porto-azygos shunts is reported to be 25%, compared to 75% porto-caval shunts⁽²⁾. However, the exact termination of a shunt is sometimes difficult to assess with ultrasonography due to its intrathoracic or dorsal location, a distended stomach, or motion of the diaphragm. Furthermore, the termination cannot always be visualised during surgery; in those cases, a porto-azygos shunt is assumed when the shunt traverses the dorsal part of the diaphragm and is located next to the oesophagus⁽²⁾. This can lead to misinterpretation

of the shunt anatomy. Computed tomographic angiography (CTA) is the gold standard for the evaluation of the portal circulation in humans and provides excellent imaging of shunt anatomy⁽¹⁾. Recently this technique was used to study the morphology of extrahepatic shunts in dogs in more detail. It was hypothesised that the morphology depends on differences in preferential flow due to valves in specific portal veins, but this needs to be confirmed⁽³⁾. Four subtypes appear to be responsible for 94% of EH shunts: spleno-caval (34%), left gastro-phrenic (22%), right gastric-caval (21%) and left gastro-azygos (16%). Another five subtypes are described but these are seen less commonly: left gastro-caval, left colic-caval, right gastro-phrenic, right gastro-azygos and complex spleno-phrenic and azygos⁽³⁾.

Intrahepatic shunts are divided into left-sided, central and right-sided shunts. Because we perform surgery of IH shunts only after visualization with CTA nowadays, high-quality images of the anatomy are available. Remarkable is the variation in diameter, length and course of intrahepatic shunts. Interesting is also that in several predisposed breeds different subtypes of intrahepatic shunts are found, as was reported in extrahepatic shunts⁽²⁾; for example, in Bernese mountain dogs and in Golden retrievers all 3 subtypes are seen. In contrast, in Irish Wolfhounds only left-sided shunt were noticed (unpublished data).

Although the development of CPSS appears to be hereditary, little is known about the pathogenesis of shunts. Before birth, numerous non-functional communications may be present between the vitelline system (forming the extrahepatic portal veins and the hepatic caudal vena cava) and the cardinal (forming systemic abdominal veins such as de azygos vein and prehepatic caudal vena cava). Extrahepatic shunts to the azygos vein or the prehepatic vena cava arise when such a communication becomes functional⁽¹⁾. Other explanations are an erroneous anastomosis of the prehepatic caudal vena cava to a vitelline vein or the anatomical proximity of involved veins in the embryo⁽³⁾. Left-sided intrahepatic shunts are probably the result of persistence of the ductus venosus. Normally this structure closes completely within a few weeks after birth⁽¹⁾. Right and central intrahepatic shunts may be caused by abnormal sinusoid development or a disturbed formation of the ductus venosus, but the exact formation of these shunts also remains unknown.

Although anatomically different, subtypes of shunts presumably have similar causative genes because in predisposed breeds often different shunt types co-exist. The age at first diagnosis or the severity of clinical signs in dogs with different subtypes may vary, for example dogs with porto-azygos shunt are significantly older when diagnosed than dogs with porto-caval shunts ⁽²⁾. Therefore, dogs with certain subtypes may reproduce before the diagnosis is made and spread causative genes in the population. More knowledge regarding the genetic background of shunts and the effects of the causative gene defects on the embryonic development of the liver and the abdominal veins, may elucidate the cause of the anatomical variation in canine shunts.

References

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