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Abdominal Ultrasound Level 2 Mini Series

Session Three: Portosystemic shunt evaluation

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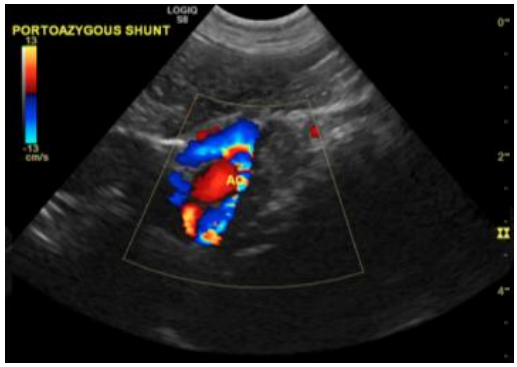


Session 3: Ultrasonography of the Portosystemic Shunt

Normal portal flow

The normal portal venous system transports blood from the intestines, spleen, stomach and pancreas to the liver. Within the liver, this blood passes through the hepatic sinusoids and eventually enters the hepatic veins and drains into the caudal vena cava (CVC).

What is a portosystemic shunt (PSS)?

A PSS is a vascular anomaly resulting from the presence of an abnormal vessel or vessels that connect the main portal vein or a smaller portal tributary and the CVC or another systemic vein such as the azygous vein. Under normal conditions, portal blood flows through the hepatic sinusoids before entering the systemic venous system. A shunt allows portal blood to bypass the hepatic sinusoids and enter the systemic venous system directly. Shunts are usually identified in young animals although occasionally, the volume of shunted blood is relatively small resulting in some cases remaining undetected for years. This occurs more commonly in animals with congenital extra-hepatic shunts. PSSs can be broadly divided into primary (congenital) and secondary (acquired) shunts.

Congenital PSSs

Congenital shunts take the form of a single (or occasionally double) macroscopic vessel. They result either from the persistence of a foetal vessel that has failed to close normally in the days after birth or due to the development of an abnormal connection between the embryonic cardinal and vitelline venous systems. In the embryo, the vitelline system forms the portal vein and its tributaries (i.e. the gastrosplenic vein, gastroduodenal vein and cranial and caudal mesenteric veins) and the cardinal system forms the systemic abdominal veins (i.e. the abdominal CVC, azygous vein, renal and gonadal veins). There should be no direct functional communication between the two systems in the normal animal. Congenital PSSs are further categorised according to whether the abnormal vessel is located inside the liver (intrahepatic) or outside the liver (extra-hepatic).

Congenital Intra-hepatic PSSs (IHPSS)

Intra-hepatic shunts are more common in large breeds of dog. They are more likely to cause clinical signs earlier in life and as such, affected animals are often only a few months old at time of diagnosis. IHPSSs are classified into left, central and right divisional shunts.

Left divisional shunts are the most common type of intra-hepatic shunt. They arise from the left branch of the portal vein and although their exact morphology varies between animals, they typically form a large, long looping vessel that joins the left hepatic vein towards the cranial aspect of the liver. In animals with a left-sided intra-hepatic shunt, the right portal branch is often very small and may even be absent. Studies have shown that the morphology of a left-divisional shunt is consistent with a patent ductus venosus. In the foetus, the ductus venosus enables oxygenated placental blood within the left umbilical vein to bypass the foetal liver and enter the CVC directly and should normally close within a few days following birth. Irish Wolfhounds have a hereditary breed predisposition to the development of left-divisional shunts.

Central divisional shunts are usually straight and occur in the region of the right medial liver lobe where they form a foramen-type communication between the intra-hepatic portal vein & ventral aspect of the CVC. They can be difficult to identify ultrasonographically and colour Doppler is often required to confirm the presence of blood flow from the portal vein directly into the CVC. Old English Sheep dogs,

Australian Cattle dogs, Labrador and Golden Retrievers are predisposed to central divisional shunts. Left and right divisional shunts also occur in Labrador and Golden Retrievers.

Right divisional shunts originate from the right intra-hepatic portal branch and pass through the right side of the liver. They often take the form of a long tortuous vessel that mirrors a left-divisional shunt but enters the right hepatic vein or CVC.

Congenital Extra-hepatic PSSs (EHPSS)

Congenital extra-hepatic shunts are most commonly diagnosed in small breeds of dog, particularly the Shih Tzu, Miniature Schnauzer, Yorkshire Terrier, Miniature Poodle, Pug and Maltese. Of the pure breed varieties of cat, Himalayans and Persians are predisposed to congenital PSSs. In cats, congenital extra-hepatic shunts are more common than intra-hepatic shunts and typically arise from the left gastric vein.

As the name suggests, congenital extra-hepatic shunts occur outside the liver between the portal vein or more commonly, a tributary of the portal vein and either the CVC itself or a systemic vein that subsequently drains into the CVC. Less commonly, shunts can also drain into the azygous vein. Traditionally, these shunts have been described as being either portocaval or portoazygous shunts however research has shown that this method of naming shunts is too simplistic. A recent series of articles by Parry, A. and White, R. have suggested that it is more appropriate to name a shunt according to the portal vessel from which it originates and the systemic venous vessel into which it terminates. For example, like cats, extra-hepatic shunts in dogs most commonly originate from the left gastric vein. These shunts can enter various systemic venous vessels including the left phrenic vein (left gastro-phrenic shunts), the pre- or post-hepatic CVC (left gastro-caval shunts) and the azygous vein (left gastro-azygous shunts). An in-depth discussion of all of the various possible permutations of extra-hepatic shunts is beyond the scope of this discussion and interested readers are referred to the references listed at the end.

Microvascular dysplasia

A third form of congenital PSS is microvascular dysplasia, now known as hepatic portal venous hypoplasia. In this form of portosystemic shunting, shunting of portal blood occurs within the liver at the microscopic level such that portal blood flows directly into hepatic venules and bypasses the hepatic sinusoids. When this condition occurs alone, there is no macroscopic shunt present although it is possible for an individual animal to have both microvascular dysplasia and a macroscopic shunt. Animals with microvascular dysplasia may have a liver of normal size and do not develop portal hypertension.

The second form of hepatic portal venous hypoplasia is known as idiopathic non-cirrhotic portal hypertension (INCPH). This is rare and thought to represent a more severe form of portal hypoplasia and can give rise to portal hypertension and the development of secondary acquired shunts.

Secondary (acquired) extra-hepatic shunts (AEHSs)

AEHSs typically present as multiple small vessels that form anastomoses between portal vein tributaries and systemic veins and arise as a result of sustained portal hypertension. It is important to realise that these vessels are normally present but non-functioning (i.e. collapsed) in the healthy animal. In animals that develop chronic portal hypertension, these pre-existing vessels open-up and become functional thereby providing a means by which portal blood can bypass the liver and enter the systemic circulation.

Portal hypertension can be pre-hepatic (i.e. affecting the pre-hepatic portal vein), hepatic or post-hepatic (involving the large hepatic veins, CVC or right side of the heart). Hepatic cirrhosis is one of the most common causes of portal hypertension in dogs. Since cirrhosis is more common in the older dog, we often associate the presence of acquired PSSs with older animals. However, young animals may also be affected (albeit uncommonly) due to the presence of conditions such as INCPH, arterioportal fistulae and juvenile hepatic fibrosis. Acquired PSS vessels vary in location but are commonly located around the spleen and in the retroperitoneal space particularly in the region of the left kidney. Occasionally, a single large dilated vessel is identified that runs from the splenic vein to the left renal or gonadal vein or directly into the CVC and is known as a splenosystemic shunt.

Imaging an animal with a suspected portosystemic shunt

There are many imaging modalities available to demonstrate the presence of a PSS including scintigraphy, mesenteric portography, magnetic resonance angiography, computed tomography angiography (CTA) and ultrasound. Of these, ultrasound and CTA are the most commonly used imaging modalities in primary and referral practice respectively.

CTA is superior to abdominal ultrasonography for the detection and characterisation of PSSs. Unlike ultrasound, shadowing from gas within the gastrointestinal tract and the lungs do not present an issue with CTA. CTA is also more useful for surgical planning since it provides an overall picture of the vessel within the abdomen whereas with ultrasound, it is only possible to view a relatively small portion of the abdomen at any given time. That said, ultrasound has the advantage of being readily available in first opinion practice, is non-invasive and has been reported to be highly sensitive for the detection of PSSs. However, the ability to detect PSSs on ultrasound is highly dependent on the skill and experience of the ultrasonographer and the quality of the ultrasound equipment being used. The choice of imaging modality will therefore depend on both availability and clinician expertise.

Hunting for a PSS on ultrasound can be a time-consuming task and patience and perseverance are required in addition to a good working knowledge of the abdominal vascular anatomy, which is considered a pre-requisite. Make sure you allow sufficient uninterrupted time to perform an ultrasound examination. It is usually preferable to have the results of routine haematology and biochemistry blood work and bile acid stimulation tests prior to ultrasound.

Secondary signs of a Portosystemic Shunt

A definitive diagnosis depends on the identification of the abnormal shunting vessel. It can be helpful to perform a routine abdominal ultrasound prior searching for the shunt itself. The three most common changes to look out for are microhepatica, renomegaly and urolithiasis. According to a study by D'Anjou et al. 2004, finding a combination of small liver, large kidneys and urolithiasis had a positive predictive value of 100% for the presence of a congenital PSS in their study population of 85 dogs and 17 cats. However, only around one third of animals with a congenital shunt had all three.

Microhepatica is present in most dogs with a congenital shunt. The liver has a dual blood supply with 20-30% of blood flow to the liver arriving via the hepatic artery and the remaining 70-80% arriving via the portal vein. An animal with a shunt has a significant reduction in blood flow to the liver resulting in reduced delivery of hepatotrophic factors to the liver and an under-developed, small liver. Whilst microhepatica is present in most dogs with a congenital macroscopic PSS, a small liver is only present in around half of dogs with microvascular dysplasia alone. Cats are more likely than dogs to have a liver of normal size despite the presence of a PSS and microhepatica is reported to occur in only around 20% of cats with a congenital shunt. The hepatic artery often becomes enlarged as a compensatory mechanism to maintain blood supply to the liver.

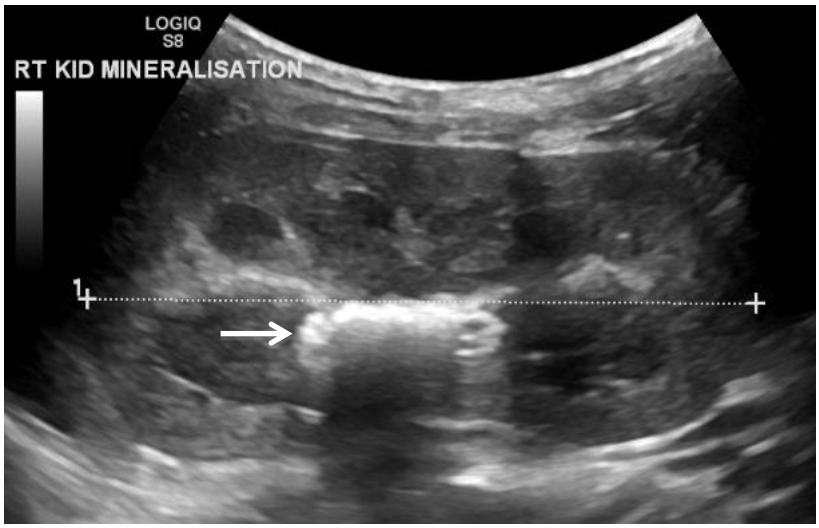
Assessment of liver size using ultrasound is a subjective process. Sometimes a small liver can appear as little more than a relatively thin slither of hepatic tissue caudal to the diaphragm. In such cases, the gall bladder will often appear to be disproportionately large relative to the volume of surrounding liver tissue. Cranial abdominal organs such as the stomach and the spleen will be located much closer than normal to the diaphragm in animals with marked microhepatica. Take care not to over-interpret the normal liver in deep-chested breeds of dog, which will naturally lie well within the costal arch.

Attempts have been made in the dog to correlate linear measurements of the liver made during ultrasound, with liver volume in order to provide an objective means of assessing liver size. However, the authors of the paper found these to be of little value in predicting actual liver weight.

A further finding in animals with a congenital PSS is reduced conspicuity of intrahepatic portal vasculature due to flow diversion and consequent hypoperfusion of the liver. Again, this is a subjective finding and relies on the sonographer having a good grasp of the normal appearance of the hepatic vasculature.

Renomegaly occurs in some animals with a congenital shunt due to an increase in glomerular filtration rate and renal volume. In cats, renomegaly secondary to a PSS is less common than in dogs although the reason for this is uncertain.

Urolithiasis: Animals of both species with a shunt are more likely to form ammonium urate stones either in the kidneys or urinary bladder. Urate calculi are usually radiolucent but they can combine with other elements such as magnesium to become mildly radiopaque.



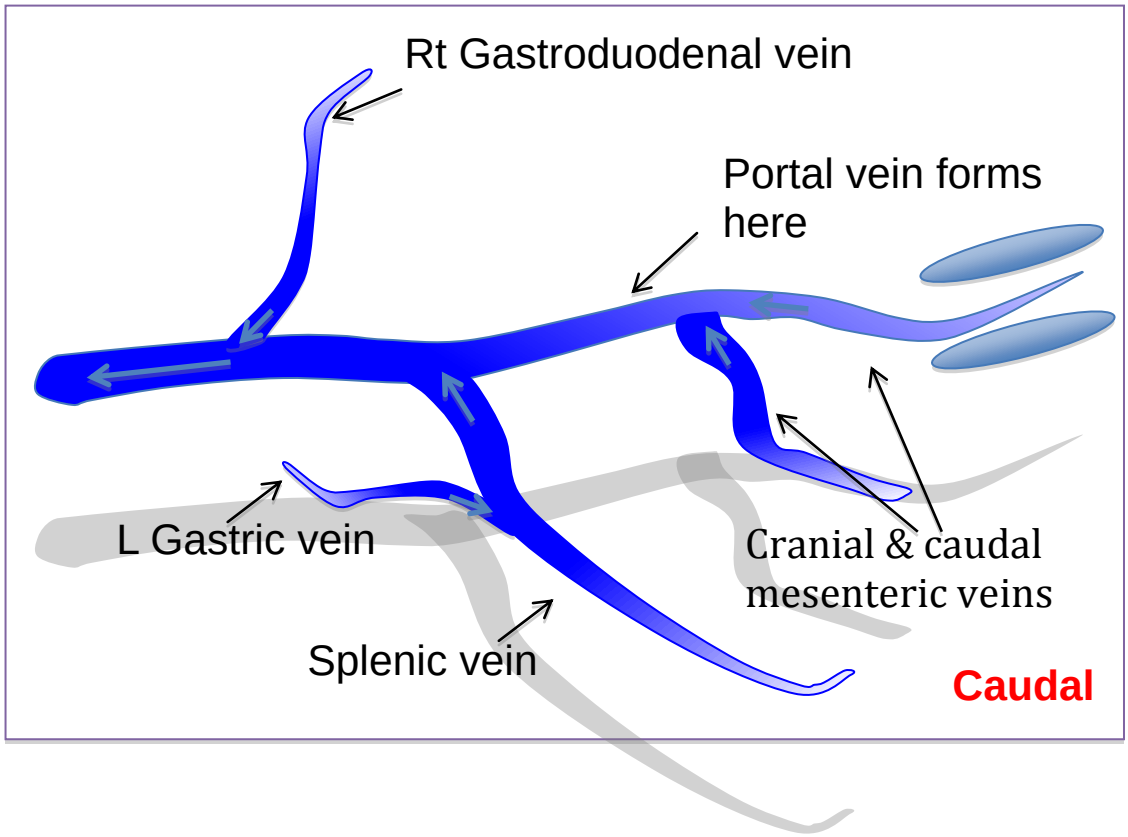
Nephrolithiasis (arrow) in a dog with a congenital intra-hepatic portosystemic shunt. Note the echogenic interface associated with distal acoustic shadowing.

Peritoneal fluid is rarely a feature of congenital shunts but is common in dogs (but not cats) with acquired PSSs arising secondary to portal hypertension. This is discussed in more detail later.

Normal Portal Vein Anatomy

As mentioned previously, you will need to start with a good working knowledge of the normal anatomy of the portal vein.

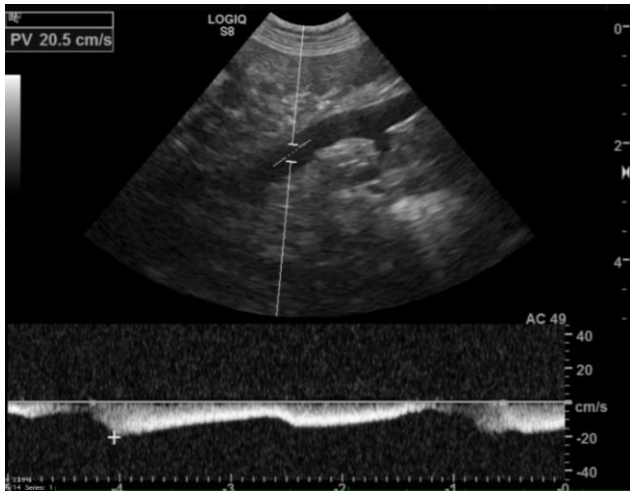
Schematic of the extra-hepatic portion of the normal portal vein:



The normal portal vein forms in the mid abdomen from the confluence of the cranial and caudal mesenteric veins. A short distance cranial to this, the gastrosplenic vein enters the main portal vein from the left. The gastrosplenic vein is formed from the confluence of the left gastric vein and the splenic vein. Further cranially, the gastroduodenal vein is the final tributary to enter the main portal vein and does do from the right side. The right gastric vein drains into the gastroduodenal vein just before the latter joins the portal vein. The portal vein then enters the liver at the porta hepatis and becomes intra-hepatic. At the porta hepatis, the portal vein is located ventral to and slightly to the left of the CVC. The aorta is the most dorsal of the three major abdominal vessels and lies just to the left of midline (and the portal vein) and is dorsal to both the portal vein and CVC. Once in the liver, the portal vein gives off a short right portal branch, which supplies the right lateral liver lobe and caudate process of the caudate lobe. The left branch is much longer and supplies the remaining lobes.

Normal Portal Vein Flow characteristics

Normal portal flow is hepatopetal i.e. towards the liver. Flow speed values vary slightly depending on which reference you read. D'anjou et al. (2004) reported a mean portal flow speed of 17.8 +/- 4.4cms^{-1} in 10 normal dogs and 17.1cm/s (range 9.7-18.1) in five unsedated cats. Lamb et al. (1994) reported normal portal flow speed in dogs to be 12-17cms^{-1}. A good rule of thumb is that mean portal flow is usually between around 10cms^{-1} and 20cms^{-1} in the normal animal.



Normal portal flow in a 6 year old Daschund being scanned due to a perineal hernia.

Portal flow in the healthy dogs is usually smooth and uniform (non-pulsatile) or varies slightly with breathing due to changes in the position of the cursor within the vessel. The portal vein is connected to the intestinal capillaries caudally and hepatic sinusoids cranially and hence is not exposed to the systolic and diastolic pressures of the systemic circulation.

Intra-hepatic Shunt Identification

Clearly, a definitive diagnosis relies on identifying the shunting vessel. Although the portal vein can be followed cranially in some dogs using a midline approach, it is often more helpful to approach a potential shunt from a left or right intercostal window. Intrahepatic shunt configuration is variable. As a general rule, left and right divisional intra-hepatic shunts tend to form relatively long vessels that deviate laterally before coursing medially to enter the left or right hepatic veins respectively, which then drain into the hepatic CVC. Central intrahepatic shunts typically form a window-like opening between the intrahepatic portal vein and CVC and colour Doppler is vital for their identification.

Under normal circumstances, flow within the CVC is cranially-directed and variable (i.e. periodic) depending on the pressure within the right atrium and changes in intra-thoracic and intra-abdominal pressure due to respiration. In the presence of a shunt, the CVC cranial to the point of shunt entry will often appear dilated due to increased flow of blood from the shunting vessel. There is usually also evidence of marked focal turbulence within the CVC at the entry point of the shunt.

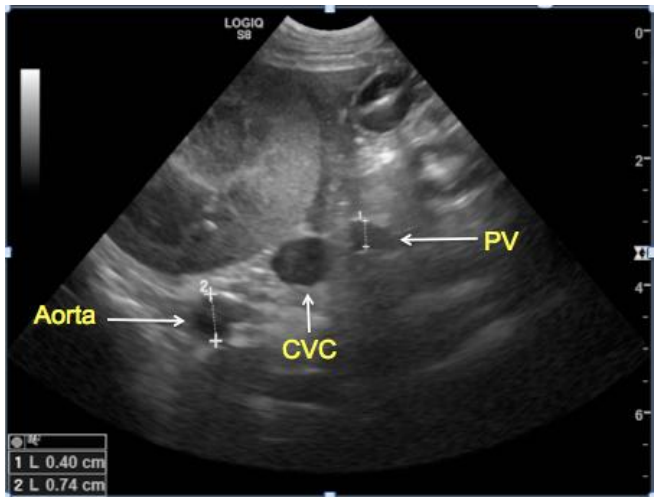
Portal flow characteristics in the presence of an IHPSS

When an IHPSS is present, there is reduced resistance to flow within the portal vein and as a result, flow speeds can exceed >20cms^{-1}. Portal flow may also be variable due to the abnormal direct connection with the systemic circulation. Not all animals with an IHPSS demonstrate pulsatility of the portal vein and some normal animals can demonstrate this (reasons not known).

Extra-hepatic Shunt Identification

Comparison of portal vein and aortic diameters can help to determine the likelihood that an extra-hepatic PSS is present. Measurements should be performed at the porta hepatis using a right cranial intercostal approach, whereby it is usually possible to produce an image of the CVC, portal vein and aorta all in the transverse plane simultaneously. Since the diameter of the aorta varies with the cardiac cycle, several frames should be reviewed and the aorta measured at its maximum diameter. The normal portal vein: aortic diameter ratio is >0.65 to 0.8. A ratio of the portal vein and aortic diameters $\leq$

0.65, is highly suggestive of an EHPSS. The reason for this is that the presence of an extra-hepatic shunt causes blood to be diverted away from the cranial portion of the portal system. Whilst idiopathic non-cirrhotic portal hypertension (portal vein hypoplasia) can also result in a small portal vein at the porta hepatis, this condition occurs much less commonly than EHPSSs.



Comparison of aortic and portal vein diameter in the cranial abdomen.

A PV : Ao >0.8 excludes an EHPSS but an intrahepatic shunt, no shunt and portal hypertension are all possible. Portal vein diameter can also be compared with the CVC diameter however it is easy to accidentally compress the CVC and hence comparison with the aorta may be more reliable. If portal vein diameter is measured caudal to the origin of an extra-hepatic shunt it may well be normal. Take care not to accidentally measure the shunt itself, a portal tributary or the hepatic artery (the hepatic artery is often dilated in animals with a congenital shunt).

Extra-hepatic portocaval shunts usually terminate in either the pre-hepatic CVC at the level of the epiploic foramen or the post-hepatic CVC. Therefore, it can be helpful to start by looking for focal turbulence in CVC at these two locations using Colour Doppler. If turbulence is identified, it may also be possible to see the anomalous vessel entering the CVC, which can then be traced in a retrograde fashion.

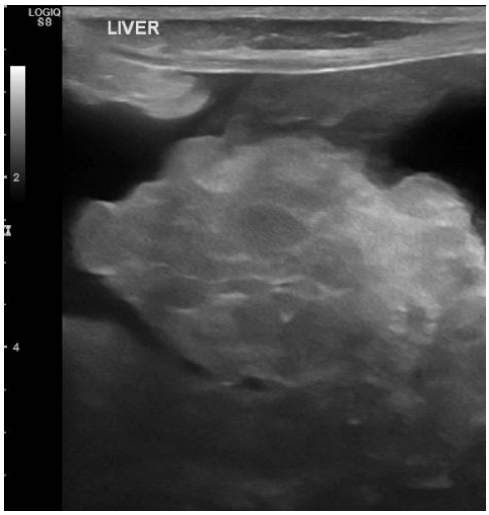
An alternative approach is to evaluate the direction of flow in the portal vein and its tributaries using Colour Doppler. Since no vessels originate from the portal vein, flow within its tributaries should always be directed into (towards) the portal vein. Flow away from the portal vein is highly suggestive of the presence of an extra-hepatic shunt (rarely, it can also be seen with severe portal hypertension). With the dog (or cat) in left lateral recumbency and the transducer on the dorsal aspect of the right mid-to cranial abdomen, start by identifying the confluence of the cranial and caudal mesenteric veins ventromedial to the descending duodenum. This marks the start of the portal vein. A short distance cranially, the gastrosplenic vein should be visible in the distal portion of the field of view entering the main portal vein. The gastrosplenic vein should be smaller in diameter than the portal vein. Assess the direction of flow in the gastrosplenic vein using Colour Doppler. Normal flow is directed towards the portal vein and from a right lateral approach, flow should be directed towards the transducer. Following the portal vein further cranially, the gastroduodenual vein should be visible in the near field draining into the portal vein. Colour flow Doppler should confirm that flow is away from the transducer (i.e. towards the portal vein) in this vessel. Further cranially, the main portal vein can be seen entering the porta hepatis.

Portal flow characteristics in the presence of an EHPSS

When an EHPSS is present, portal flow caudal to shunt is often increased in speed (>20cms^{-1}) due to reduced resistance to flow. Within the portal vein cranial to the shunt, flow speed is usually reduced (<10cms^{-1}) and may even be reversed (hepatofugal) due to the diversion of blood into the shunting vessel. In theory therefore, this abrupt change in portal flow speed can be used to determine the origin of shunt however the small size of the portal vein cranial to the origin of a shunt can make assessment of flow speed in this portion of the vein, challenging. Care should be taken not to accidentally measure flow in the shunting vessel itself.

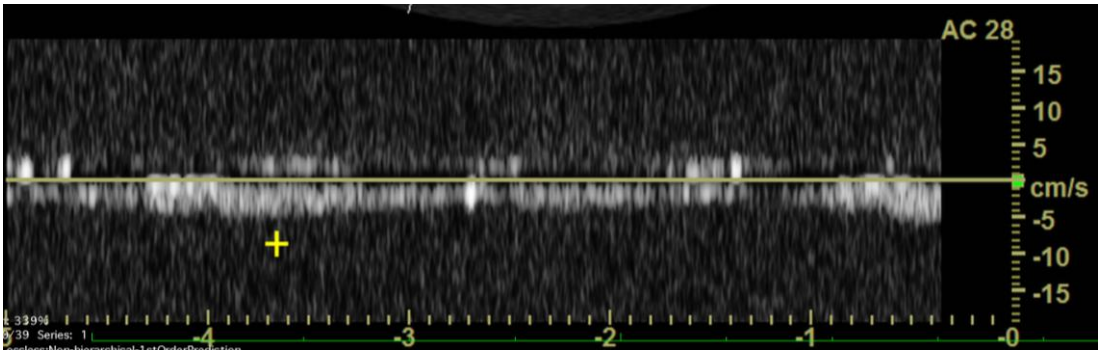
Acquired Portosystemic shunts

As previously mentioned, acquired PSSs develop due to sustained portal vein hypertension. Portal hypertension can develop in the presence of a number of conditions including cirrhosis, non-cirrhotic portal hypoplasia, arterioportal fistulae, external compression of the extra-hepatic portal vein (e.g. by enlarged hepatic lymph nodes) and portal vein thrombosis.



Confirmed cirrhosis of the liver in a 10 year old dog. Note the nodular, irregular appearance of the liver and the presence of free anechoic peritoneal fluid.

A cirrhotic liver will usually appear small and nodular. Ascites is a common concurrent finding in animals with severe chronic portal hypertension as is oedema of the pancreas, gall bladder wall and gastric wall. The portal vein may be normal to large in diameter at the porta hepatis and portal flow may be reduced in speed (<10cm/s) and even hepatofugal.

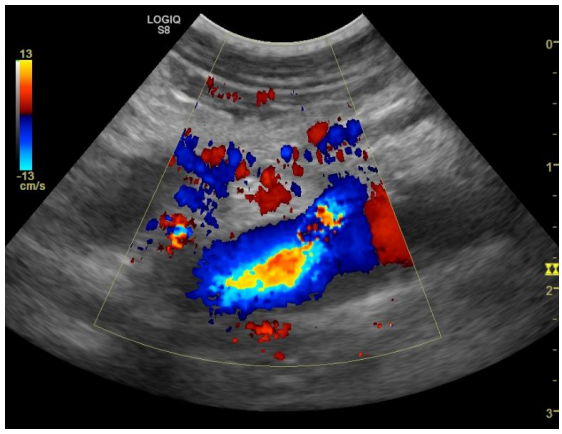


Doppler spectrum in an 8 year old X-breed dog with portal hypertension secondary to cirrhosis. Maximum flow speed within the portal vein is around 5cms^{-1}.

Secondary acquired shunts usually present as multiple, very tiny, tortuous vessels. Whilst they can develop at multiple sites throughout the abdomen, the best place to look is usually in the retroperitoneal space in the region of the left kidney. It is usually necessary to apply colour Doppler to appreciate the extent of shunting present. It is not usually possible to follow these vessels from their origin to their termination.

The easiest way to detect a splenosystemic shunt is to look for a relatively large-diameter vessel entering either the left renal vein or the CVC adjacent to the renal vein. Despite the fact that splenosystemic shunts are solitary, they are still classed as acquired shunts.

It is normally recommended that ultrasound should be performed in the fasted animal to reduce intestinal luminal gas, which can obscure anatomy. However, in animals where multiple acquired shunts are expected but not seen, repeating the ultrasound exam post-prandially may make it easier to spot these small vessels, which dilate in response to the increased portal venous blood flow following a meal.



4 year old Yorkshire Terrier with multiple acquired shunts in the region of the left kidney secondary to portal hypertension due to underlying non-cirrhotic portal hypertension.

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