Hepatic arteriovenous malformation in a puppy: Dual treatment approach involving interventional radiology and open surgery

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History

Prince, a 6-month-old, male entire Border Collie presented to the internal medicine team with a three-day history of lethargy, pacing and head-pressing. After physical and neurological examinations, blood analyses were performed looking for a metabolic cause for the neurological signs. There was a marked elevation of serum bile acids and ammonia suggestive of hepatic encephalopathy as a cause of neurological signs.

An initial abdominal ultrasound was highly suspicious for a large intrahepatic portocaval shunt and a thrombus in one of the branches of the shunt. Computed tomodensitometry (CT) with angiography of the abdomen was performed to better understand this hepatic vascular anomaly prior to potential correction. The CT scan identified an arteriovenous malformation, as opposed to a portocaval shunt. This rarely described malformation consisted of a communication between the hepatic branch of the celiac artery and left intrahepatic portal vein (Figure 1). In addition, acquired gastrophrenic, splenorenal and mesenteric portal collateral shunting vessels were visible, most likely secondary to the portal venous hypertension caused by the arteriovenous malformation. Peritoneal effusion was present which was also attributed to portal hypertension. The presence of a portal vein thrombus was confirmed on CT scan presumably due to blood turbulence and potential damage to the vascular...
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Prince was admitted to our intensive care unit and his neurological signs were initially stabilized with IV fluid therapy, lactulose enemas, intravenous amoxicillin/clavulanic acid and nutrition with a hepatic (low protein/soy based) diet. Intravenous omeprazole was prescribed due to the known risk of gastrointestinal bleeding in dogs with portal hypertension. Antithrombotic therapy with oral apixaban and clopidogrel was introduced due to the finding of portal thrombus.

Hepatic arteriovenous malformation is a rare condition and the treatment has traditionally been surgical (Chanoit et al. Vet Surg 2007). However, an interventional radiology approach aiming at embolising the anomalous vessel(s) was also described in dogs (Chanoit et al. Vet Surg 2007, Case et al. JAVMA 2017).

After a week of medical management that failed to normalise the plasma ammonia, Prince underwent an interventional radiology procedure where 80 to 95 % of the abnormal arterioportal communication could embolised (Figure 2). Multiple coils were placed in order to block the blood flow through the arteriovenous malformation. Due to rare nature of this condition an expert in human interventional radiology worked with us.

Prince recovered well from the procedure and the ammonia values the following day had returned to normal for the first time. Postoperative abdominal ultrasound that day revealed the presence of air in the gall bladder wall, raising concerns about gallbladder wall necrosis as a complication of vascular embolization with involvement of the celiac artery. Persistent hepatofugal portal blood flow (abnormal flow pattern where the portal venous flow is going away from the liver rather than towards the liver) was still present as previously documented on preoperative ultrasound. However, the velocity of this flow was reduced compared to that prior to the intervention.

Based on these ultrasonographic findings, Prince was scheduled for a laparotomy two days after the interventional radiology procedure. The plan was to perform a cholecystectomy and attempt to occlude the visible residual arterioportal shunting vessel(s). A standard cholecystectomy was performed without complication. The ventral surface of the left lateral lobe was covered by a thick tortuous network of anomalous arterial vessels emptying in a large dilated portal vascular structure with turbulent blood flow (Figure 3 – page 30). The anomalous arterial vessels could be digitally compressed and were clipped using several large hemoclips. This led to immediate reduction of the size of the dilated portal vascular structure and apparent resolution of the turbulent blood flow within this structure. Following this second procedure Prince’s abdominal ultrasound revealed a hepatopetal portal blood flow for the first time (normal flow pattern where the portal venous blood is flowing towards the liver).

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The plasma ammonia remained within normal limits following this second procedure and Prince was discharged 5 days later. We have rechecked him regularly since then and were very pleased with his progress. The owners do not report any more signs compatible with hepatic encephalopathy. They actually mentioned that he is a completely different puppy since his anomaly was corrected, very energetic and playful (Figure 4). His nutritional management plan was recently changed from a hepatic diet to a non-protein restricted high-quality diet, which he is tolerating well.

**Discussion**

There is minimal information on hepatic arteriovenous malformation in veterinary medicine and the majority of the information is extracted from human medicine. There are differences between an arteriovenous fistulae and arteriovenous malformations, despite that both are high-flow vascular anomalies. An arteriovenous fistula is a single communication between an artery and vein. An example is a patent ductus arteriosus. An arteriovenous malformation consists of multiple small communications involving a “nidus” of vessels and can be more difficult to identify.

In human medicine, the treatment of hepatic arteriovenous malformations consists of embolization of the nidus.

The treatment of small communications and not the nidus can lead to worsening of the lesion and make further treatments even more difficult. The treatment is often palliative and not curative, and in the majority of the cases multiple treatments are needed.

Hepatic arteriovenous malformations in canine patients are thought to be congenital in origin. In contrast, arteriovenous fistulae can be congenital or acquired. An arteriovenous malformation is a rare condition, which appears to affect dogs more often than cats and it is typically diagnosed in young patients. Typically, there are several vessels connecting the hepatic arterial bed and the hepatic portal venous bed. Due to the pressure gradient between the hepatic artery and the portal vein, the blood is flowing from the hepatic artery towards the portal vein leading to an increase in portal venous pressure.

As a consequence, multiple acquired extrahepatic portosystemic shunts can develop as well as signs of gastrointestinal haemorrhage and signs of hepatic encephalopathy. Ascites is reported in 75% of cases of arteriovenous malformation.
in dogs, which is not the case with portosystemic shunts. In the 25% of dogs that do not have ascites, a hepatofugal portal blood flow is expected. Other clinical signs reported include diarrhoea, vomiting, stunted growth, lethargy, and heart murmurs.

In canine patients, correction of arteriovenous malformation can be attempted by ligation of the nutrient artery, liver lobectomy or transarterial embolization (which can be done with glue or application of coils). Liver lobectomy and transarterial embolization seem to be the options associated with better outcome.

Surgery can be challenging, as the majority of the arteriovenous malformations are located in the right and central hepatic divisions and often involve the gallbladder vascularisation or caudal vena cava. Surgical complications can include haemorrhage, portal hypertension, systemic hypotension, bradycardia, and portal or mesenteric vein thrombus formation. In dogs undergoing surgical therapy alone, perioperative survival rates were 77% and long-term outcome was fair or good in 38% and 57% of cases, respectively. Overall 75% of dogs continue to require dietary or medical management of clinical signs due to persistence of acquired portosystemic shunting (Chanoit et al. Vet Surg 2007).

Transcatheter therapies typically involve cyanoacrylate glue or coils. There is currently minimal information about the use of these techniques in dogs. One publication describes that 4 dogs treated by glue embolization alone were alive without clinical signs in a follow-up period of 9 to 17 months (Chanoit et al. Vet Surg 2007). A more recent article reported a successful outcome in a dog treated by combined coil and glue embolization without requirement for open surgery (Case et al. JAVMA 2017).

The originality of our case lies on the fact that a dual staged approach was used for treatment. It involved coil embolization of the vascular nidus first to attenuate the degree of arteriovenous shunting, followed by complete surgical ligation of the remaining patent vessels in open surgery. It is our impression, based on the extent of Prince’s malformation, that it would not have been amenable for complete surgical ligation without prior attenuation with coil embolization.

Prince is a lovely dog and we are delighted to have been able to work together within our multidisciplinary team, alongside a medical colleague, to be able to deliver a positive outcome for both him and his owners.

**References**


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