Invited lecture/Review

Portosystemic Shunts in Cats

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Abstract:
The portosystemic shunt (PSS) is an abnormal communicating vessel between the portal and systemic vasculature. The liver is underdeveloped due to decreased blood flow. The PSS may be congenital (intrahepatic or congenital) or acquired (extrahepatic). Ammonia and intestinal toxins are not cleared in the liver, causing various clinical signs of hepatic encephalopathy. Medical treatment is aimed at minimising clinical signs and stabilising the cat prior to surgical treatment. Surgical treatment is currently the method of choice; however, the best method of occluding the shunts has yet to be found. Surgical treatment involves complete ligation of the shunt in cases where the portal vasculature is adequately developed, but more commonly the surgery aims to occlude the shunt gradually with a cellophane banding or ameroid ring. The cellophane may not produce the same fibrotic response as in dogs, so clinical signs often improve, but the cat still requires medication. In general, the prognosis after surgery in cats is not as good as in dogs.

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1. Introduction

The portosystemic shunt (PSS) is an abnormal communicating vessel between the portal and systemic vasculature that causes decreased blood flow to the liver, preventing the liver developing normally (Tivers and Lipscomb, 2011). In normal animals, blood from the stomach, intestine, pancreas and spleen is transported to the liver via the portal vein to perfuse the liver, after that the blood enters the hepatic veins, and then the caudal vena cava (Tobias, 2003; Tivers and Lipscomb, 2011). The liver performs many important functions, including the metabolism of toxic or harmful substances absorbed from the gastrointestinal (GI) tract. Proteins that enter the GI tract are degraded by anaerobic and coliform bacteria via urea to ammonia, which is transported to the liver and converted to urea.
Ammonia in cats with PSS is not metabolised by the liver but enters the systemic circulation directly, which is why ammonia is elevated in blood (Tivers and Lipscomb, 2011). Due to decreased oxygenated and nutrient-rich blood supply through the portal vein, the liver is underdeveloped in cats with PSS. Usually, the liver is small (hypoplastic) and has inadequate function. High levels of waste products (such as ammonia and intestinal toxins) are transported directly into the systemic circulation via the shunting vessel (Hottinger et al., 1995), causing various clinical signs related to hepatic encephalopathy and the central nervous system (Lipscomb et al., 2007; Tivers and Lipscomb, 2011). Hepatic encephalopathy can be triggered by high-protein meals, GI bleeding, and anaesthetics (Tivers and Lipscomb, 2011).
PSS can be congenital or acquired. A congenital PSS is an abnormal communicating vessel between the portal and systemic vasculature, they may be inta- or extrahepatic; whereas acquired PSS develop secondary to underlying liver disease (Lipscomb et al., 2007; Tivers and Lipscomb, 2011). The most common forms of PSS in cats are extrahepatic (73–100%) (Birchard and Sherding, 1992; Levy et al., 1995; Lipscomb et al., 2007). Congenital PSS are rare in cats with an incidence of 2.5 per 10,000 cats (Levy et al., 1995). Although Hunt (2004a) stated that there is no association between breed and the type of shunt found in cats, other studies suggest that extrahepatic PSS most commonly affects domestic shorthair cats, followed by Persian, British shorthair, ragdoll, domestic longhair, Birman, British blue, and Tonkinese, whereas intrahepatic PSS most commonly affects Siamese cats. In terms of gender male cats are most affected (Lipscomb et al., 2007).

2. Clinical manifestation

Cats with PSS present with nonspecific clinical signs that are typically episodic and worsen after feeding (Lipscomb et al., 2007; Tivers and Lipscomb, 2011). Cats may present with hypersalivation and neurological signs such as ataxia, head pressing, strange behaviour, lethargy, aggression, tremors, blindness, and seizures. Cats may be small, the body condition score (BCS) may be low, but even a normal BCS does not rule out PSS. Some cats have a copper-coloured irises (**Figure 1**). Gastrointestinal signs such as anorexia, vomiting and diarrhoea have been noted, and urinary signs (dysuria, haematuria) due to ammonium urate urolithiasis may also be observed (Lipscomb et al., 2007). Due to impaired metabolism in the liver, cats may require a longer recovery time after anaesthesia (Tivers and Lipscomb, 2011).

Figure 1. Copper coloured irises in a cat with PSS.

3. Diagnosis

The diagnosis of PSS can be made based on laboratory findings such as routine haematology and biochemistry, urinalysis – ammonium biurate crystals (Kyles, et al.2002), abdominal ultrasound, computed tomography (CT), MRI, and nuclear scintigraphy (Tivers and Lipscomb, 2011). Biochemical changes, such as decreased urea concentrations in blood can be seen in cats with PSS when more than 70% of liver function have been lost and decreased creatinine in blood can also be seen in cats with low muscle mass (Kyles et al., 2002; Tobias, 2003). Alkaline phosphatase (ALP) and alanine aminotransferase (ALT) in blood are normal or may be moderately elevated (cite). Albumins are usually not decreased as it is common in dogs (Kyles et al., 2002).
CT angiography is the gold standard for diagnosing PSS in cats and provides highly detailed information about shunt morphology and the hepatic vasculature (Zwingenberger, 2009). The final diagnosis is made at the time of surgery (Tivers and Lipscomb, 2011). Hepatic function can be assessed by bile acids and ammonia blood testing. Postprandial bile acids in blood have a sensitivity of 100% and fasting bile acids in blood have a specificity of 84% and both have the best predictive values for the diagnosis of PSS in cats (Center et al., 1995). Fasting ammonia, with a sensitivity of 83% and a specificity of 86% is elevated in most cats with PSS (Ruland et. al., 2010).

4. Treatment options

Medical treatment of cats with PSS is indicated to minimise clinical signs, to stabilise the cat before surgical treatment, or when surgery is not possible because of the location of the shunt. Medical treatment is important to decrease the absorption of ammonia and toxic products from the gastrointestinal tract and to prevent hepatic encephalopathy (Broome et al., 2004) and consists of lactulose, low-protein diet, oral antibiotics, and anticonvulsants. Oral administration of antibiotics (ampicillin 10 - 20 mg/kg/8h or metronidazole 10 mg/kg/12h) reduces the gastrointestinal population of ammonia-producing anaerobic and Gram-negative bacteria. Lactulose (0.5 - 2 ml/8 - 12h, orally) reduces ammonia production and absorption. To lower protein intake, cats should be fed a commercial diet with low to moderate protein content (Tivers and Lipscomb, 2011). For cats, which have higher protein requirements than dogs, commercial liver support diets with moderate amounts of high-quality protein are recommended. Vegetable protein sources are not recommended for cats (Lidbury et al., 2016).
Cats suffering from seizures should receive phenobarbitone (1 - 4 mg/kg orally/12 h), propofol infusion (0.05-0.4 mg/kg/min) and levetiracetam (20 mg/kg/intravenously (IV)). Acid-base status, dehydration, glucose, coagulation profiles, and electrolytes in blood should be corrected as needed (Tivers and Lipscomb, 2011; Tonge, 2021). Vitamin K should be administered 1 - 2 days before surgery when coagulation profiles are prolonged (Self, 2016). If gastrointestinal bleeding is suspected, gastroprotective drugs should be administered (Lidbury et al., 2016).

5. Anaesthesia

Cats should be fasting 3 - 4 hours before anaesthesia, and blood glucose levels should be monitored throughout the perioperative period. Methadone 0.1 - 0.2 mg/kg intramuscularly (IM) or IV as premedication provides intraoperative as well as postoperative analgesia (Tonge, 2021). Remifentanyl 5 - 40 μg/kg/h IV or fentanyl 5 - 8 μg/kg/h IV should be administered as a constant rate infusion during surgery (Day, 2013; Self, 2016). If sedation with an opioid alone is not sufficient, a low dose of medetomidine 0.5-3 μg/kg IM or dexmedetomidine 0.5 - 1.5 μg/kg IV or IM can be administered. Alpha-2 receptor agonists such as medetomidine and dexmedetomidine are reliable sedatives that reduce the drug doses required for induction and maintenance of anaesthesia and also contribute to analgesia (Lemke, 2004; Murrell and Hellebrekers, 2005). Acepromazine should not be used in patients with PSS because it can lead to an excessively long duration of action and prolonged hypotension and has no reversal agent (Murrell, 2016).
Propofol or alfaxalone are commonly used for IV induction of anaesthesia (Self, 2016). Maintenance of anaesthesia with isoflurane or sevoflurane reduces the risk of potential drug accumulation (Day, 2013; Self, 2016). Isoflurane or sevoflurane can cause dose-dependent hypotension (Bernard et al., 1992). During anaesthesia, IV Ringer's lactate should

be administered at a rate of 3 ml/kg/h (Davis et al., 2013), and in the event of hypotension, a bolus of hydroxyethyl starch (3 ml/kg) should be administered and repeated after 15 minutes if needed (Day, 2013). Monitoring during anaesthesia is important to assess depth of anaesthesia and cardiorespiratory function.

This includes pulse oximetry, end-tidal carbon dioxide concentration, isoflurane concentration, non-invasive arterial blood pressure measurement with Doppler, electrocardiogram, and temperature (Day, 2013). It is also important to monitor palpebral reflexes, eye position, pulse rate and quality, and respiratory rate and depth (Tonge, 2021).
Repeated administration of opioids after surgery should be based on objective pain scales, with an established intervention value rather than predetermined dosing intervals (Tonge, 2021). If seizures occur during recovery, they should be treated with an IV infusion of propofol (Heldman et al.,1999) and/or with levetiracetam (20 mg/kgIV) or phenobarbitone (2 - 3 mg/kg IV) (Tivers and Lipscomb, 2011). The use of benzodiazepines is not recommended in patients with PSS (Self, 2016). Non-steroidal anti-inflammatory drugs are contraindicated due to liver disease. Medical treatment should be continued for at least two weeks after surgery.

6. Surgical treatment

Surgical treatment consists of complete or partial closure of the shunt. Some authors (Youmans and Hunt, 1998) recommend slow occlusion to reduce the risk of life-threatening portal hypertension. This can be done with an ameroid ring or cellophane band (Vogt et al., 1996; Hunt et al., 2004), although clinical signs may persist (Hottinger et al., 1995). If the portal vasculature is sufficiently developed, ligation of the shunt with nonabsorbable suture is also possible (Burton and White, 2001; Lipscomb et al., 2007; Tivers and Lipscomb, 2011).

7. Prognostic factors

The prognosis for cats after surgical treatment has been reported to be moderate to poor, with long-term survival rates varying from 56 - 85%. In cats that survive the postoperative period and do not develop seizures, prognosis is good (Valiente et al., 2020). Death may occur in the intraoperative or early postoperative period and is caused by portal hypertension, complications of anaesthesia, portal vein thrombosis, seizures (Hottinger et al., 1995; Vogt et al., 1996; Hunt et al., 2004b), hypothermia, cardiorespiratory arrest (Hunt et al., 2004b), or euthanasia due to presumed portal vein atresia and inability to occlude the shunt (Lipscomb et al., 2007). Hunt et al. found (2004b) that after closure of shunts with cellophane band in cats, shunt closure may fail, or multiple acquired shunts may occur. If a shunt vessel is only partially closed clinical signs may still be present, although possibly to a lesser degree (Hottinger et al., 1995). A good indicator of shunt attenuation is bile acid stimulation, which correlates well with clinical outcome (Valiente et al., 2020).

8. Conclusion

Clinical signs in cats with PSS may be nonspecific and intermittent, so shunts are often not recognised early in the animal's life. Whenever a cat is presented with hypersalivation, copper-coloured irises, neurological signs, and prolonged recovery time after anaesthesia for elective surgery, PSS should be suspected. Prognosis after surgical occlusion is better compared with medical treatment alone but generally worse than in dogs. If the shunt remains patent after surgery, further occlusion may be attempted.

Conflicts of Interest: The authors declare no conflict of interest.

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