Nursing the Portosystemic Shunt Patient
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Abstract
This lecture takes a detailed look at nursing portosystemic shunt patients. It discusses presenting clinical signs of portosystemic shunt, and the diagnosis of it, and the medical and surgical options for treatment. It then focuses on surgical treatment in detail, going through surgical preparation and anaesthesia, the surgery itself, and post operative nursing care.

Learning outcomes
- Understanding of the different clinical signs of portosystemic shunt
- Understanding of the different options for diagnosis and treatment
- Understanding of portosystemic shunt surgery and port operative care.

Course Notes

Introduction
A portosystemic shunt (PSS) is any vascular anomaly that allows blood from the hepatic portal circulation to bypass the liver and be delivered directly into the systemic circulation.

Extrahepatic shunts (EHPSS) are vascular anomalies located outside the hepatic parenchyma. Intrahepatic shunts (IHPSS) are located within the liver. Extrahepatic shunts may be congenital, usually a signal anomalous vessel or acquired, often multiple small vessels. EHPSS account for nearly 63% of single shunts in dogs; they also occur in cats (Fossum 2007).

Presenting Signs
Blood bypasses the liver in a PSS, which results in the following:

- Poor hepatic development and atrophy are thought to be due to the reduced delivery of hepatotrophic substances from the portal vein.
- Decreased protein production
- Reticuloendothelial dysfunction
- Altered fat and protein metabolism
(Fossum 2007)
The reduced portal vein blood flow to the hepatic parenchyma impairs the liver’s ability to detoxify digested metabolites. The severity of clinical signs is thought to be related to the volume of blood bypassing the liver. The clinical signs that occur as a consequence of the shunting blood are chronic gastrointestinal signs, lower urinary tract signs, coagulopathies and stunted growth (Berent and Tobias 2009). The clinical syndrome of hepatic encephalopathy can be associated with altered CNS function (Fossum 2007).

Signs of lower urinary tract disease are common due to decreased hepatic urea production leading to increased urinary ammonia excretion. This predisposes to the development of ammonium urate crystalluria and urolithiasis. Acute lower urinary tract obstruction may occur as a consequence. Patients may also be polydipsic and polyuric as a result of a poor medullary concentration gradient (Berent and Tobias 2009).

**Diagnosis**

**Imaging**

The diagnosis of PSS is confirmed by diagnostic imaging. Methods include:

- **Ultrasound** – Is widely available and therefore has become a commonly used method to identify PSS. Ultrasound diagnosis is dependent upon operator experience. With EHPSS, overlying bowel may obscure the shunt, but a small liver with few detectable hepatic or portal veins may be noted.
- **Radiographs including contrast procedures** - The bladder and renal pelvises should be assessed for calculi, these usually are radiolucent and difficult to observe on abdominal radiographs. Microhepatica and enlarged kidneys may also be evident however radiographs are more effective in identifying these.
- **Computer tomography (CT)** - is the gold standard for evaluating the portal venous system in people (Ettinger 2010). This method of imaging is becoming increasingly more popular in animals but requires sedation or an anaesthetic.
- **Magnetic Resonance Imaging (MRI)** - can be used to view the abnormal vasculature. Contrast medium is injected intravenously and then a series of images are taken to create a 3D image (Bruehschwein et al 2010).
- **Scintigraphy** - Rectal scintigraphy is less commonly used to diagnose PSS.

**Laboratory findings**

Haematology, serum biochemical and urine analysis of animals with PSS may show variable abnormalities, but dogs can have a congenital PSS without any abnormalities on complete blood count or serum biochemistry panel.

Fasting (12 hours) and 2 hour postprandial serum bile acids are most widely used tests for evaluating liver function in animals with PSS. By measuring these levels this entire circuit is evaluated (Berent and Tobias 2009).

Blood ammonia levels are also increased due to decreased hepatic ammonia detoxification. Coagulation profiles often show prolonged clotting times as most of the clotting factors are synthesised in the liver.
Medical vs Surgical

Medical management should be instigated prior to surgery, this usually includes:

- Antibiotics - decrease GI bacteria numbers, allowing for decreases in ammonia production.
- Lactulose - Traps ammonia in the form or ammonium, and it also increases gut transit time, therefore reducing bacterial metabolite absorption.
- Diet - highly digestible, high biological value of protein, fatty acids, vitamins and minerals to meet the minimum requirement for the individual.

(Berent and Tobias 2009)

These management techniques are an attempt to optimise the patient’s condition and reduce the likelihood of postoperative conditions. Surgery is the treatment of choice for most animals with PSS because hepatic function may continue to deteriorate as long as most blood is shunted away from the liver. Patients that are cachectic, encephalopathic or unstable should be managed medically until they can tolerate the stress of anaesthesia and surgery. All patients may benefit on longer medical management to increase body condition score (BCS) prior to surgery.

Anaesthesia monitoring techniques and considerations:

<table>
<thead>
<tr>
<th>Procedure</th>
<th>Used for</th>
</tr>
</thead>
<tbody>
<tr>
<td>Peripheral Intravenous</td>
<td>Allows the administration of intravenous fluids and induction of anaesthesia</td>
</tr>
<tr>
<td>Central Venous Catheter</td>
<td>Allows measurement of CVP during shunt ligation and access to blood sample collection for TP, PCV and blood glucose</td>
</tr>
<tr>
<td>Arterial Catheter</td>
<td>Allow rapid measurement of arterial blood pressure although. Access to blood sample collection for blood gas analysis, TP, PCV and blood glucose</td>
</tr>
<tr>
<td>Doppler</td>
<td>Technique can be used if at arterial catheter cannot be placed.</td>
</tr>
<tr>
<td>ECG</td>
<td>To monitor heart rate and rhythm during surgery</td>
</tr>
<tr>
<td>SP02</td>
<td>To monitor oxygen saturation during surgery</td>
</tr>
<tr>
<td>etCO2</td>
<td>To monitor end tidal CO2 during surgery</td>
</tr>
</tbody>
</table>

Adjusted from (Seymour and Duke-Novakowski 2007)

Patients with PSS are often hypersensitive even at light levels of anaesthesia and therefore a balanced anaesthetic technique is advantageous. Hypoglycaemia can result due to impaired ability
of the liver to synthesise glucose and reduced hepatic glycogen stores. Therefore, blood glucose levels should be monitored throughout anaesthesia.

Care should be taken to prevent hypothermia as these patients are often small and they have a large surface area:volume ratio and readily lose core body heat due to open abdominal cavity and the surgical preparation solution used.

**Surgery options:**

The preferred treatment for PSS is complete/partially shunt attenuation. Surgical attenuation is intended to redirect the portal blood flow through the liver to promote normalisation of the hepatic structure and function (Karla et al 2006).

An ideal method of surgical treatment would be a single quick, inexpensive procedure that resulted in gradual attenuation without the risk of portal venous hypertension. Most will not tolerate complete occlusion of shunts therefore gradual attenuation is preferred to reduce the risk of post-operative complication. Successful surgical attenuation of shunts can lead to a complete clinical recovery without the need for long-term medical or dietary treatment.

Once the anomalous vessel is identified it will be temporarily occluded with plastic tubing (or) and nylon tape, then portovenogram performed to ensure the correct vessel is occluded. The surgery can be continued with the attenuation method of the surgeon’s preference.

Ameroid constrictors and cellophane bands are the most common attenuation techniques used due to suture technique as it should avoid the need for a second procedure. Ameroids and preferred physiologically as the liver has chance to adapt to blood flow. They are less likely to cause portal hypertension making them a convenient option, whereby less monitoring is required (Fossum 2007).

Pre and post ligation portograms are used to assess the portohepatic blood supply in animals undergoing suture attenuation of PSS. Complete ligation of the shunt at the first procedure-if using suture is not often possible and so a second procedure is required 4 to 6 weeks later to fully occlude the shunt. Complete attenuation of portosystemic shunts is recommended to achieve resolution of clinical signs and improve long-term survival. Acute complications for the nurse to observe include:-

- Haemorrhage
- Hypoglycaemia
- Hypothermia
- Portal hypertension
- Prolonged recovery
- Seizures

**Post General Anaesthesia**

Hypothermia during recovery is a common problem and can be minimised by recovering patients in an incubator.
Blood pressure should be monitored every 15–30 minutes. These patients are often hypotensive (MAP <60mmHg). Colloids can be given if the patient is hypotensive and does not respond to crystalloids. Fluid therapy should be continued until the patient is eating and drinking.

Blood glucose should be monitored hourly until the patient is awake.

Due to reduced liver function drugs may be metabolised more slowly than normal and so dosing intervals and/or amounts may need to be adjusted. The patient should be pain scored hourly using the Glasgow Composite Pain Score (Murrell, J 2008). If a score of 4 or more is demonstrated then the analgesia plan should be reevaluated. Non-steroidal anti-inflammatory drugs should be avoided if possible due to the risk of GI ulceration associated with portal hypertension.

General nursing care should be provided to meet the patient’s individual needs such as turning recumbent patients every 2 hours, lubricating eyes and ensuring the head and neck are extended once extubated.

**First 24 hours**

Vital signs should be monitored every 2–4 hours.

The analgesia plan can be reevaluated depending on the pain score. Analgesia can be staged down from methadone to buprenorphine. The patient should be pain scored when the analgesia is expected to wear off.

An in-house PCV/total solids, electrolytes and acid base should be performed daily.

The central venous catheter is usually kept in situ for the first 24 hours or until the patient is eating and drinking with enthusiasm. The central line should be checked twice daily for signs of infection, inflammation or migration. It should be kept clean and handled with the strictest hygiene.

Mentation should be monitored every 4 hours. If mentation is altered then there may be a concern that the patient is becoming encephalopathic due to elevated ammonia within the bloodstream. This can be checked via a laboratory test and the patient can be assessed using the Modified Glasgow Coma Score (Platt 2009). Treatment for HE is often aggressive and requires lactulose enemas, oral lactulose and a switch from oral antibiotics to intravenous preparations. In severe cases the patient may seizure and require anticonvulsants such as levitaracetam and even induction of a propofol coma. As a standard policy we issue each PSS patient with a seizure sheet which indicates the course of treatment should the patient seizure.

The abdominal circumference can be measured with a tape measure to monitor for portal hypertension. Signs of portal hypertension include ascites, pain, melaena and systemic hypotension (Mehl 2009).

Most patients will already be on a protein restricted diet. As these patients are often small and prone to hypoglycaemia, it is important to encourage eating as soon as possible.

The medical management that the patient was receiving prior to surgery should be continued. This should help reduce the risk of hepatic encephalopathy.
Patients with liver disease can have coagulation disorders, and so they should be carefully monitored for signs of bleeding, particularly if liver biopsies have been taken.

The surgical wound should have a light adhesive dressing applied for the first 24 hours until a fibrin seal has formed. If there is strike through to the dressing then it should be replaced.

Urine output should be monitored by noting on the hospitalisation record when the patient voids their bladder. The absence of urination may indicate acute kidney injury.

48 – 72 hours

If the patient is eating and drinking then intravenous fluid therapy can be discontinued. If the patient is comfortable on the pain score then analgesia can be terminated.

The surgical wound should be monitored daily for signs of inflammation or infection. The central line can be removed if it has not been yet. At this point the patient should be fit for discharge or if hospitalisation is required until the owners can collect their pet then they can be hospitalised in a normal ward area. Medical management is usually continued for at least 4 weeks post-op when repeat laboratory tests will be scheduled.