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Approach to urethral obstruction in cats. Part 1: presentation and stabilisation

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Background: Urethral obstruction is one of the most commonly encountered emergencies in feline medicine, with a wide range of clinical signs, from dysuria to peri-arrest. Such patients are some of the most challenging cases in feline medicine with several factors to consider; however, they can be equally rewarding.

Aim of the article: This is the first article in a three-part series. The aim of this article is to summarise a stepwise approach from presentation through to diagnosis of urethral obstruction. The second article will focus on urethral catheterisation, and immediate postcatheterisation management, while the third will focus on home management to prevent recurrent obstruction, and on addressing underlying feline idiopathic cystitis.

DESPITE being a common emergency in general practice, feline urethral obstruction remains a complex condition to manage, with several controversies to consider. With their longer and narrower urethra, this is predominantly a condition of male cats, although it can occasionally be seen in female cats too.

Feline urethral obstruction usually occurs as part of feline lower urinary tract disease (FLUTD); this is the umbrella term for all causes of cystitis and associated presentations in cats (Gerber and others 2005). The most common causes of obstruction are urethral plugs (composed of proteinaceous debris in which crystals can become trapped) and urethral spasm, both of which are common consequences of feline idiopathic cystitis (FIC), also known as 'stress cystitis', and cause 65 to 90 per cent of cases of urethral obstruction. Other causes include urolithiasis (10 to 35 per cent of cases), of which calcium oxalate and struvite are both common types of stones. Slater and others (2020) also found strictures and urethral trauma to be present in 15 per cent of cases. Blood clots occur occasionally, as can neoplasia of the urethra, prostate or bladder. Very rare cases of feline urethral obstruction are associated with significant perineal or intrapelvic pathology or trauma.

KEY LEARNING OUTCOMES

After reading this article, you should:

- ☐ Have an understanding of the common causes of urethral obstruction;
- ☐ Be able to perform the triage examination;
- ☐ Be able to decide the most useful emergency diagnostics to perform;
- ☐ Be able to formulate a fluid plan for collapsed cats;
- ☐ Be able to address hyperkalaemia in obstructed cats.

Triage

When the cat arrives with a history of unproductive straining, one of the first questions to be asked is: how stable is the patient? This is because there is a spectrum of presentations, ranging from mild cystitis-like signs to peri-arrest. A focused evaluation is needed to answer this question, with the key components of the triage examination in dysuric cats being:

- mentation and whether this is appropriate for the environment
- mucous membrane colour
- capillary refill time
- heart rate and rhythm
- pulse quality and whether it is synchronous with heart rate
- respiratory rate and lung sounds
- abdominal palpation for bladder size
- rectal temperature could be considered if it won't cause excessive stress, as axillary temperatures are less likely to be reliable in a hypovolaemic cat.

These parameters provide rapid information on the stability of the major body systems (cardiovascular, respiratory and CNS) that are likely to result in acute decompensation. If these vitals do not provide cause for concern, then the full clinical examination can be completed.

It should be noted that although bradycardia can be associated with hyperkalaemia, it is also a common presentation in cats with any form of shock.

Additional information to be ascertained from a secondary examination once the patient has been identified as stable includes:

- hydration status
- abdominal discomfort
- weight, taking into account the bladder volume
- rectal examination if collapsed, if not then once the cat is sedated/anaesthetised.

Emergency diagnostics

As a matter of urgency an intravenous (IV) catheter, from which blood can be withdrawn, should be placed and secured. As these patients typically have circulatory collapse in the later stages, it may be necessary to use several 1 ml syringes with only slight negative pressure applied. Depending on the in-house analysers available, having a store of precoated lithium heparin syringes, such as those used for arterial samples, can be helpful when there is sluggish blood flow from the catheter.

Emergency bloodwork is the diagnostic equivalent of physical triage, where the key parameters that are likely to change your management in the acute period are rapidly assessed. At our institution, emergency bloodwork would encompass the following:

- packed cell volume
- total solids
- glucose
- lactate
- electrolytes
- blood smear evaluation.

If residual blood is available, priority should be given to evaluating renal parameters and ideally, ionised calcium concentration.

Blood pressure should be obtained, with Doppler measurement being the most reflective of true systolic blood pressure in cats (Waddell and Brown 2015). Although the measured pressure might not always be an accurate reflection of body-wide perfusion, it can be useful to provide an objective baseline to help with decision making. Blood pressure can be determined during the initial diagnostic evaluation, before blood sampling, as this may worsen situational hypertension ('white coat effect') in stable patients. This phenomenon is unlikely to cause interference in collapsed cats, as sympathetic drive should already be maximal. A systolic blood pressure below 90 mmHg is consistent with a mean arterial pressure below 65 mmHg, at which point renal perfusion is inconsistent and fluid resuscitation is urgently required. Hypotensive cats carry a more guarded prognosis.

A point of care ultrasound (POCUS) examination is very useful in these patients. It can be performed without the need for extensive clipping of the coat and does not require advanced equipment or skill. Cover the probe with a glove or cling film containing ultrasound gel, then spray surgical spirit or water into the parted hair to achieve a decent acoustic window while also protecting the probe.

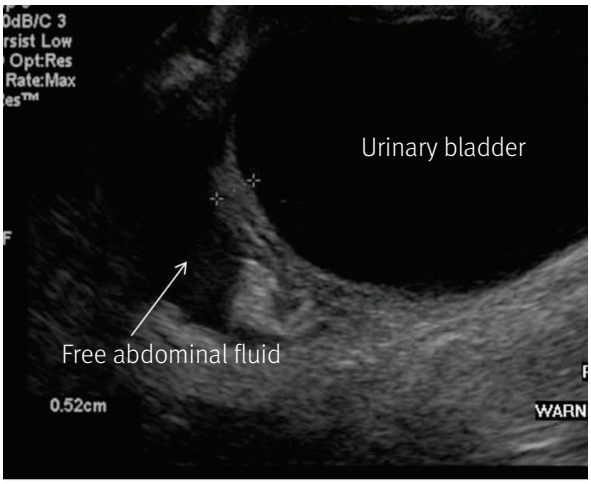

Fig 1: This depicts a point of care ultrasound (POCUS) image, focused on the apex of the bladder. There is a triangle of free fluid cranial to the bladder, where it most often accumulates if small in volume

The aim of the POCUS is not to evaluate the urogenital system in great detail, but instead to evaluate whether there is free fluid within the abdomen, and whether the urinary bladder appears intact; be aware that the presence of a bladder on imaging does not exclude a tear or leak. In feline urethral obstruction, there is often a small amount of free abdominal fluid (Fig 1) and, although not typically detected, larger volumes can be seen with rupture along the urinary tract and subsequent uroabdomen.

To aid identification of small volumes of free abdominal fluid, POCUS involves focusing on four main areas in the abdomen:

- between the liver lobes and diaphragm
- between the spleen and left kidney
- between the liver and right kidney
- around the bladder.

Although most patients presenting with urethral obstruction are typically young male cats that are otherwise healthy and capable of tolerating fluid administration, in older patients or those with pre-existing heart disease, it is sensible to evaluate the heart via ultrasound before giving IV fluids. Scanning the cat from the right side of the chest, over the apex beat, allows the left atrial to aorta (LA:Ao) ratio to be measured (Fig 2). Cats with urethral obstruction are usually hypovolaemic, so the ratio will be low, whereas cats with ongoing heart disease are likely to have left atrial enlargement, so the ratio may be increased. A ratio greater than 1.6 is consistent with enlargement of the left atrium, and caution should be exercised with any fluid therapy.

With the triage examination performed and these diagnostic results obtained, a wealth of information

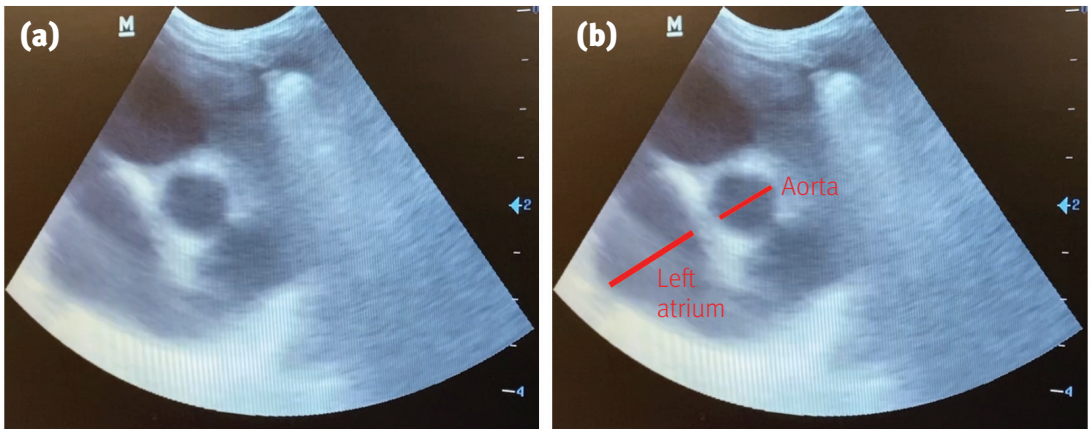

Fig 2: (a) This point of care ultrasound (POCUS) image is a right parasternal short axis view of the heart. (b) The aorta is in the centre of the image and the left atrium at the 6 to 8 o'clock position

is rapidly available to aid in clinical decision making.

ECG can be used in these patients if available. This has the benefit of allowing a continued recording of the heart rate. It can also be useful in evaluating for hyperkalaemia, discussed later in this article. Although consistent ECG findings and bradycardia can be a specific finding for hyperkalaemia, their absence does not exclude it and should not be solely relied upon.

Stabilisation

Analgesia

One of the first aspects to be addressed is the provision of analgesia. Cats with urethral obstruction are incredibly painful secondary to severe bladder distension; some may also have pain from preceding inflammation of the bladder wall. Interpreting clinical parameters and making fluid therapy decisions is difficult when pain is stimulating the sympathetic drive, manifesting as raised blood pressure, heart and respiratory rates.

Cats with urethral obstruction are often unstable when first presented, making it difficult to know whether surgical intervention may be needed later. Therefore, if possible, a full opioid, which should maximally stimulate and bind to the receptors, is advised. Methadone (0.2 mg/kg given IV) is a reasonable option, which leaves potential for increasing the dose, if necessary. While buprenorphine is very effective in cats, the concern is that if the analgesia proves inadequate and a more potent opioid becomes necessary, the agonist/antagonist effect may impede the full opioid effect for six to eight hours.

Maropitant may have some visceral analgesic properties and could be considered in conjunction with the proven analgesics above, particularly as these patients are likely to experience nausea associated with the shock state.

NSAID medications, while often a mainstay in these patients when stable, should be avoided at presentation. Renal and gastrointestinal perfusion is likely to be inconsistent due to hypovolaemia, and if anaesthesia were to become necessary then vasodilation and worsening hypotension is a possibility, which could add further insult to already injured kidneys.

Fluid therapy

Fluid therapy is an area in which several controversies exist. Although it would seem counterintuitive to provide more fluid when there is obstruction of urine output, it is a necessity for stabilisation of the patient. The fluid can help restoration of renal perfusion and dilute the plasma potassium.

The first question is: Which fluid type should be used? An isotonic crystalloid is most often selected, and the decision is then whether to use 0.9 per cent sodium chloride or Hartmann's. The theoretical advantage of saline is that it includes no additional potassium, so it should address the hyperkalaemia more effectively. The disadvantage is that it has an acidifying effect, whereas Hartmann's is alkalinising, which is relevant in these patients because they are likely to be acidaemic. However, the evidence indicates that the type of isotonic crystalloid used has no significant difference in prognosis or the ability to correct the hyperkalaemia. The only difference was that those patients receiving Hartmann's normalised their acid-base disturbances more quickly (Cunha and others 2010). This is because improved renal perfusion allows for greater excretion of potassium into the urine, in excess of the potassium accumulated from the fluid therapy. As such, Hartmann's is typically the first-line fluid choice in our hospital.

The next question to be addressed is: what volume of fluid should be administered? This can be answered by deciding what we are trying to address. These patients are likely to be dehydrated, at least to some degree, with a loss of fluid in the interstitial space; however, this is not a major concern in the acute setting. Dehydration is not life threatening in itself but becomes so when it leads to a loss of fluid from the intravascular space, causing hypovolaemia

with resultant kidney hypoperfusion. This gives rise to azotaemia and an inability to excrete potassium. This is the problem that must be targeted first.

Administering rapid IV shock doses of fluids, which is the cat's blood volume (50 ml/kg), was historically recommended in patients with hypovolaemia and shock. However, this can result in fluid overload with associated complications, especially in cats. Fluid overload can manifest as chemosis, serous nasal discharge and pulmonary oedema or pleural effusions secondary to cardiac overload. The approach has now shifted to dividing the shock dose of fluids and giving aliquots of it over a short space of time to restore intravascular volume. Boluses for hypovolaemic cats usually consist of 5 to 10 ml/kg being administered over 10 to 15 minutes, depending on how haemodynamically stable the patient is. In those with severe hypovolaemia, larger volumes can be administered.

After each bolus, the patient is then re-evaluated for improvement in the following perfusion parameters:

- mentation
- heart rate
- pulse quality
- mucous membrane colour
- capillary refill time
- blood pressure.

Lactate can also be measured as an objective marker if available. Approximately a quarter of the fluid bolus will stay in the intravascular space, with the remainder redistributing to the interstitial space. The amount that is drawn to the extravascular space may increase with dehydration. This can manifest as an initial response to the fluid bolus, then recurrence of clinical signs of hypoperfusion after 30 to 60 minutes as the fluid leaves the intravascular space. Once the perfusion parameters have normalised, the fluid rate can be set to replace the degree of dehydration, to address ongoing losses and provide maintenance fluids. If there are no signs of dehydration, such as dry mucous membranes, prolonged skin tent or sunken eyes, then subclinical dehydration of 5 per cent can be assumed.

Hyperkalaemia

Pathophysiology

Hyperkalaemia, and its resultant cardiotoxic effect, is the immediate concern in these patients. Understanding the mechanism by which elevated potassium causes these deleterious effects can be helpful in appreciating the treatment options.

Potassium is essential for myocardial cells to generate and transmit an action potential. The action potential is generated, and the myocardial cell is depolarised by the influx of positive sodium (Na+) ions from the extracellular space to the negative inside of the cell membrane. Once the cell is depolarised, it is refractory to the generation of further action potentials. To restore negativity to the inside of the cell, and allow future action potentials to occur, the sodium-potassium-ATPase (Na-K-ATPase) pump pushes potassium ions (K+) out of the cell and the positive charge with it.

In states of hyperkalaemia, the potassium concentration across the cell membrane is no longer present to the same degree as normal. This reduces the amount of potassium that is ejected from the cell, and consequently the inner cell membrane stays too positive and remains refractory. In effect, the resting potential (or baseline cell polarity) is elevated and more positive. With the cells unable to repolarise, the clinical manifestation of this is bradycardia or atrial standstill if severe. Fig 3 summarises this process.

Diagnosis

Definitively diagnosing hyperkalaemia is reliant upon measuring serum electrolyte concentrations. The degree of increase can be summarised as:

- mild: >5.5 mmol/l
- moderate: >6.5 mmol/l
- severe: >7.5 mmol/l.

Myocardial toxicity is possible once the serum potassium concentration is above 6.0 mmol/l; however, the likelihood will vary depending on other factors, particularly serum ionised calcium concentration.

Hypocalcaemia exacerbates the effect of the hyperkalaemia by decreasing the threshold potential, meaning the initial action potential is generated easily, but repolarising the cell in the face of hyperkalaemia becomes more difficult, if not impossible. Therefore, it is important to assess blood calcium concentration in these cases. Ionised hypocalcaemia is present in 75 per cent of cats with urethral obstruction; its severity correlates with more serious cardiovascular compromise and a poorer prognosis (Drobatz and Hughes 1997). This is summarised in Fig 3i.

Surrogate markers of hyperkalaemia include bradycardia identified on clinical examination and ECG alterations. ECG findings suggestive of hyperkalaemia include sinus bradycardia, which can progress to an atrioventricular block or atrial standstill if severe. Other features of the ECG include an absence of P waves, widening of the QRS complex and the classically reported tenting of the T waves. As the hyperkalaemia becomes severe, it can present as a sine wave. Fig 4 provides an example of this, in which P waves are absent and T waves spiked.

Treatment

Treatments, doses and important notes are summarised in Table 1.

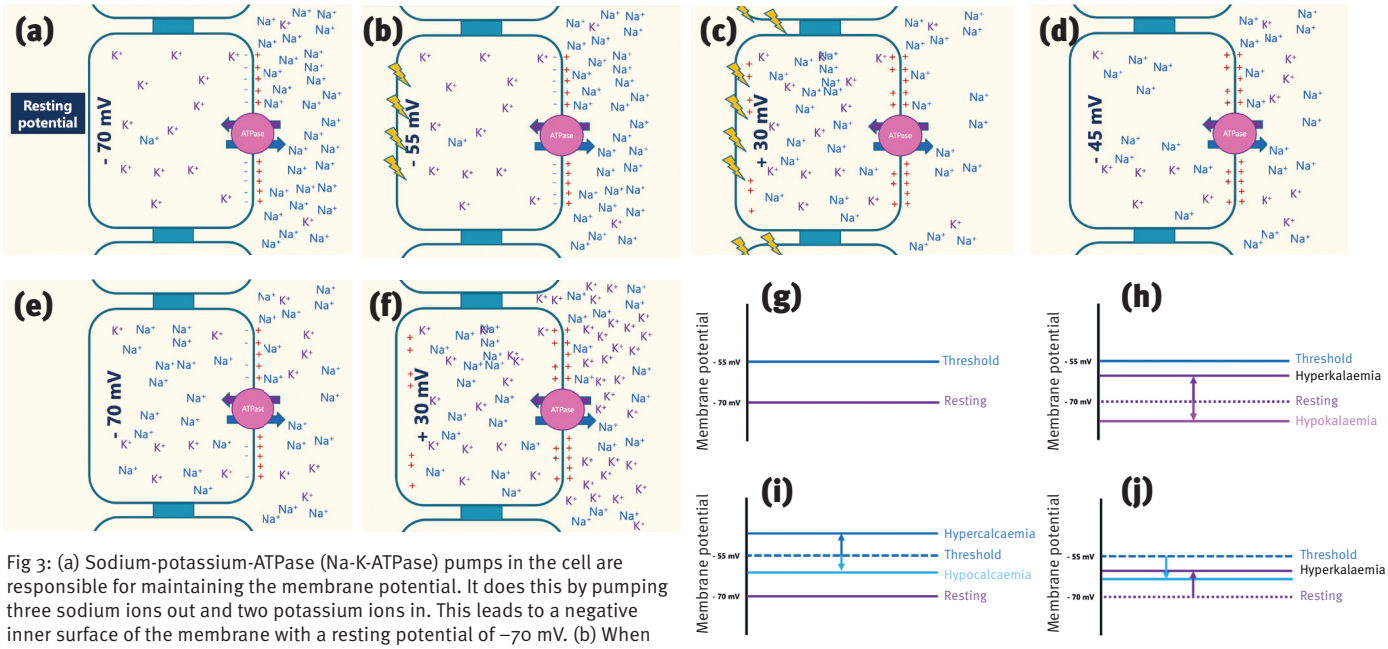

Fig 3: (a) Sodium-potassium-ATPase (Na-K-ATPase) pumps in the cell are responsible for maintaining the membrane potential. It does this by pumping three sodium ions out and two potassium ions in. This leads to a negative inner surface of the membrane with a resting potential of –70 mV. (b) When an action potential is stimulated, this causes a gradual increase in the membrane potential up to the point of –55 mV. When this point is reached (the threshold potential), a large number of sodium ions open, resulting in a large number of sodium ions entering the cell down their electrochemical gradient. (c) This results in the cell becoming depolarised to a value of approximately +30 mV. (d) To repolarise and prepare for a future depolarisation, the cell must lose positive charge rapidly. To achieve this, potassium channels open, allowing potassium ions to leave the cell down their electrochemical gradient. (e) The Na-K-ATPase pump is then activated again. The membrane potential is re-established at –70 mV. (f) In cases of hyperkalaemia, at the stage when potassium channels open (ie, step d), the electrochemical gradient is reduced with the increased circulating potassium. This prevents the negative membrane potential being re-established, thus preventing future depolarisation. (g) This depicts the difference between the resting potential, where the cell is maintained when the Na-K-ATPase pump is functional, with normal electrolyte levels. (h) Potassium concentrations within the blood have an impact on the resting potential of the membrane. With hyperkalaemia it is raised, making it easier to reach the threshold potential and trigger depolarisation. However, it is more difficult to then repolarise the cell, meaning the myocyte is then refractory to repeat action potentials. (i) Serum calcium concentration alters the threshold potential. Increasing calcium levels also increase the threshold potential, making it more difficult to depolarise. This explains the rationale for administering calcium to treat hyperkalaemia, as it restores the magnitude of difference of the resting and threshold potentials, albeit both at new points. (j) Equally, it demonstrates why those cats with ionised hypocalcaemia are at a higher risk of the deleterious effects of hyperkalaemia

Calcium gluconate

This is the first-line medication to treat life-threatening bradycardia associated with hyperkalaemia. Calcium gluconate will alter the electrical charges of the cell membrane, which increases the threshold potential, and thus redresses the balance of the resting and threshold potentials needed to restore normal electrical activity. Calcium gluconate will not alter the serum potassium concentration but will act as a cardioprotectant, providing time to establish other methods to address the hyperkalaemia.

Calcium gluconate 10 per cent is dosed at 0.5 to 1.5 ml/kg (equating to 50 to 150 mg/kg of calcium gluconate). It should be given slowly intravenously, over 20 minutes. Ideally, an ECG should be in place while the calcium is being infused, or if unavailable then monitoring the heart rate with auscultation is an alternative. Potential side effects include bradycardia and ECG changes, including sinus bradycardia, shortening of the QT interval and S-T elevation.

The beneficial effects of calcium are relatively short lived, lasting from 20 to 30 minutes, providing time to address the hyperkalaemia directly.

Fluid bolus

As discussed above, a fluid bolus will have several beneficial effects. By restoring renal perfusion and glomerular filtration, the surplus potassium can be filtered and excreted into the urine. Additionally, acidosis promotes the movement of potassium from the intracellular to extracellular space. Fluid therapy, particularly with Hartmann's solution, will address the acidaemia and cause a shift of potassium back into the intracellular space.

Fig 4: Continuous ECG, which depicts the changes encountered in a patient with clinical hyperkalaemia, including absence of a P wave and spiked T wave

Table 1: Medicines available for treating hyperkalaemia in cats

Drug	Dose	Notes
Calcium gluconate 10 per cent	0.5 to 1.5 ml/kg	•Ideally give over 20 to 30 minutes, but can be given over 5 minutes if necessary •Monitor with ECG or auscultation •Lasts for 20 mins
Glucose	0.5 g/kg if used alone for mild hyperkalaemia; 2 g per unit of insulin given as a bolus followed by a glucose infusion	•Dilute 1:4 with 0.9 per cent sodium chloride to minimise risk of phlebitis
Neutral insulin	0.25 to 0.5 iu/kg	•Glucose should be monitored for up to 24 hours after administration •Glucose constant rate infusion will likely be necessary
Terbutaline	0.01 mg/kg	•Give slowly intravenously •Tachycardia may occur •Variable efficacy •This is of theoretical benefit, with no published studies on its clinical use and would be off licence
Sodium bicarbonate	1 to 2 mEq/kg	•Rarely necessary, and other measures should be attempted first to address pH •Has numerous side effects that should be taken into account

Glucose and insulin

If the hyperkalaemia is severe, it is causing a significant bradycardia or the potassium levels have not improved with fluid therapy, then glucose and insulin is indicated. The insulin causes the glucose to be taken up into the cell, and this takes potassium ions with it, driving an intracellular shift. Glucose alone may help stimulate endogenous insulin release; however, exogenous insulin will facilitate this more rapidly. Although this will not reduce the body-wide potassium levels, it will decrease serum levels, sparing the myocardium of the deleterious effects.

Regular (soluble) insulin is dosed at 0.25 to 0.5 units/kg IV as a slow bolus.

Dextrose is then administered at 4 ml of 50 per cent dextrose per unit of insulin given. However, it should be noted that 50 per cent dextrose has high osmolarity and will cause phlebitis if given into a peripheral vein at this concentration. Therefore, it should be diluted with 0.9 per cent saline to a concentration of 1:4.

There is the risk of hypoglycaemia following the insulin administration, even when a glucose bolus is given. Frequent monitoring of the blood glucose concentration is essential, and a dextrose infusion is likely to be needed. If necessary, a 2.5 per cent dextrose infusion can be made by adding 50 ml of 50 per cent dextrose (25 g dextrose) to 1 l of fluid; this is then given as part of the fluid plan in the hours that follow.

Terbutaline

Terbutaline is a β2 agonist, which is often used as a bronchodilator in emergency situations. It also has the additional benefit of stimulating the Na-K-ATPase on cell membranes, with the resultant effect that potassium is driven intracellularly, thus decreasing the circulating hyperkalaemia. Side effects can include tachycardia by stimulating the adrenergic receptors on the myocardial cells. Rarely, severe tachycardia can result in poor cardiac filling and resultant hypotension.

Sodium bicarbonate

If the cat is severely acidotic and hyperkalaemic, then bicarbonate can be used to correct the acidosis and drive the potassium intracellularly. However, this is rarely necessary as fluid therapy alone is usually sufficient to correct this. In addition, there are side effects associated with sodium bicarbonate administration which could pose serious risks, such as ionised hypocalcaemia, hypernatraemia and central acidosis.

Cystocentesis

Should cystocentesis be performed in cases of urethral obstruction? Although the azotaemia that occurs in these cats is multifactorial, the major contribution is postrenal in origin. With the urethra obstructed, the bladder becomes excessively distended, causing back pressure via the ureters to the renal pelvis. At a microscopic level the pressure in the nephrons is markedly elevated, which opposes glomerular filtration, resulting in the potassium and uraemic toxins remaining in the circulation rather than getting into the filtrate.

The plan should be to stabilise and anaesthetise the patient as quickly as possible, so that a urinary catheter can be placed. However, in some patients, significant pressure within the bladder may confound efforts to pass a urinary catheter,

and the ongoing bladder distension and metabolic disturbances become the life-threatening issue. Cystocentesis may then be needed to relieve bladder pressure and facilitate urinary catheter placement.

Unless the cat is so severely collapsed that wriggling is unlikely, it is best to perform cystocentesis once the cat is sedated or anaesthetised. The main risks are bladder rupture, precipitating a vagal event or laceration of a vessel resulting in a haemoabdomen. Some degree of uroabdomen is always likely; however, it is unlikely to cause a problem unless the cat has a concurrent urinary tract infection. Cats with FIC are at particular risk of uroabdomen as their bladder urothelium and detrusor muscle are badly compromised by their underlying disease. Vagovagal collapse has been reported to occur in cats and can be fatal (Odunayo and others 2015). While these risks are rare they must be communicated to the owner.

If cystocentesis is to be performed, some strategies can minimise the risk of iatrogenic damage. Ideally, this is performed with two people, using the same equipment as would be required for a thoracocentesis. One person inserts the needle or butterfly catheter into the bladder, with ultrasound guidance if necessary. This person holds the needle stable and the second person attaches the butterfly catheter line, or additional tubing, to a three-way tap and syringe. With the initial person focusing on keeping the needle steady, this stops it moving unnecessarily within the bladder, preventing microtrauma or laceration of the bladder wall. The bladder should be drained until it is empty. This removes the pressure component and minimises the risk of urine leakage from the bladder. The urine can then be collected and stored for analysis and culture if indicated.

If uroabdomen does develop, while less than ideal, it is still preferable to ongoing obstruction and secondary hyperkalaemia in a cat that is unable to be catheterised. Once a urinary catheter is placed, this will keep the bladder decompressed in the hope that any bladder defect will close and heal; even sizeable tears can heal in this way when managed correctly. The abdominal effusion can be removed, if significant, by abdominocentesis to decrease the potassium load. Severe cases of uroabdomen will need to be referred for more extensive management.

One published study assessed sedation and analgesia with intermittent cystocentesis instead of urethral catheter placement where there were severe financial limitations, and there were no significant serum biochemical changes from the urethral obstruction (Cooper and others 2010). Cats had cystocentesis performed up to 10 times; four out of 15 cats developed abdominal effusion, of which three had uroabdomen and one had haemoabdomen. Therefore, complications can be severe, but this risky and suboptimal approach could be discussed with owners where funds are severely constrained.

Summary

Although urethral obstructions can be tricky cases, a stepwise and methodical approach, with the focus on emergency stabilisation, is key to ensuring these cats are stabilised enough for more definitive treatment.

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