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Welcome

This month’s *Feline Focus* covers some diverse topics including an article on the management of uroabdomens by Cecilia Stilwell, which illustrates the importance of pre-operative stabilisation. We continue our series on urinalysis by looking at urine specific gravity (USG). Measuring USG seems such a simple thing but it tells us so much about the cat’s health, so take every opportunity to use the refractometer. We complete our Keeping Cats Safe series with an article on ‘high-rise’ syndrome, a serious condition comprising multiple traumatic injuries in some cases when cats don’t land on their feet! Finally, we have a case study on diabetic ketoacidosis by Jim Littlewood, a presentation that requires considerable nursing input for a successful outcome.

Thanks so much for reading and for helping cats! Our nurse and technician membership continues to grow but we want to reach more people to help more cats — tell your colleagues and friends to sign up at icatcare.org/nurses.

Best wishes,

Sam Taylor, Veterinary Editor

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Medical management of uroabdomen in cats

Uroabdomen is the accumulation of urine within the abdominal cavity following injury to the urinary tract. This condition is most commonly caused by trauma, with the bladder being the most common site of rupture. Urine in the abdominal cavity causes azotaemia, metabolic and electrolyte derangements and cardiac disturbances, which can become life-threatening. Medical stabilisation is imperative before definitive surgical correction can be considered. The prognosis for these patients improves in the absence of concurrent injuries and with rapid stabilisation. It is therefore essential that the veterinarian and veterinary nurses and technicians work closely to identify and stabilise these patients quickly and effectively.

Uroabdomen is a medical emergency that requires rapid recognition and implementation of therapy. Uroabdomen is the accumulation of urine within the peritoneal cavity, retroperitoneal cavity or both, secondary to rupture of the urinary tract. Uroperitoneum is most commonly encountered. This condition occurs following loss of integrity to the lower urinary tract (distal ureters, bladder or proximal urethra). In comparison, uroretroperitoneum will develop following injury to the kidney or proximal ureter.1

When urine accumulates in the peritoneal or retroperitoneal cavities severe electrolyte and metabolic changes can occur. If left untreated these derangements will have harmful effects on the cardiac and renal function of the cat and can become life-threatening.

This review focuses on the common causes of uroabdomen in cats and discusses the approach to diagnosis and subsequent medical stabilisation of these cats.

Key point
Uroperitoneum is a medical emergency. The presence of urine in the abdomen causes severe electrolyte and metabolic changes which can become life-threatening if not treated rapidly.

What are the causes of uroabdomen?
There are numerous causes of uroabdomen, these can be traumatic or non-traumatic in origin (Box 1). Uroabdomen in cats is most often associated with trauma, for example blunt or vehicle trauma. In these cases, the urinary bladder is the most common site of rupture.2,3

Cecilia Stilwell graduated from the University of Glasgow in 2014. She has spent a period of time in first opinion small animal practice and is currently undertaking an internship at Dick White Referrals, UK. Cecilia has a strong interest in internal medicine, in particular nephrology and urology.

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BVMS MRCVS

Medical management of uroabdomen in cats

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Cecilia Stilwell
BVMS MRCVS
Iatrogenic urinary tract rupture is the most common non-traumatic cause of uroabdomen.\textsuperscript{3}

**Box 1: Causes of uroperitoneum**

**Trauma:**
- blunt;
- vehicles.

**Iatrogenic:**
- surgical (urogenital or abdominal);
- urethral catheterisation;
- cystocentesis;
- manual bladder expression.

**Obstruction:**
- urolith in the kidney, ureter or urethra;
- pelvic masses;
- bladder neoplasia.

**How is uroabdomen diagnosed?**
A diagnosis of uroabdomen is based on the history, clinical examination findings, laboratory evaluation and diagnostic imaging studies.

**History**
It is important to ascertain whether the cat has a history of trauma, feline lower urinary tract disease, urolithiasis, recent abdominal or urogenital surgery.

**Clinical examination**
The severity of clinical signs will depend on the duration of the uroabdomen. The possibility of uroabdomen should be considered in any patient with a history of trauma. The clinical signs include:
- lethargy;
- anorexia;
- vomiting;
- stranguria;
- haematuria;
- abdominal distension with a palpable fluid thrill;
- severe abdominal pain, which may present as aggression.\textsuperscript{1,4,5}

**Key point**
Cats in hypovolaemic shock can present with bradycardia or tachycardia.

In severe cases the cat may present in hypovolaemic shock. It is important to be aware that cats in shock may present differently to dogs. Cats can be tachycardic or bradycardic with pale mucous membranes, weak pulses and cool extremities. They may also be tachypnoeic, hypothermic and show signs of generalised weakness and/or decreased mentation.\textsuperscript{6}

**Laboratory evaluation**
All cats with a suspected uroabdomen should have blood taken for analysis. The initial tests should include a complete blood count, electrolytes, serum biochemistry and acid-base analysis.\textsuperscript{1,5,7} The most common abnormalities encountered with uroabdomen are:
- raised urea and creatinine (azotaemia);
- metabolic acidosis;
- electrolytes disturbances (mild hyponatraemia, hyperphosphataemia, hypocalcaemia and hyperkalaemia).\textsuperscript{1,4,7}

**Key point**
The presence of a palpable bladder and an ability to urinate does not exclude the possibility of a ruptured urinary tract and subsequent uroabdomen.\textsuperscript{2}
If an abdominal effusion is identified, a sample of the effusion should be collected. An abdominocentesis can be performed blind or under the guidance of ultrasound. The biochemical and cytological characteristics of this fluid should be examined. A definitive diagnosis of uroabdomen is made by comparing the creatinine and potassium levels within the serum to the abdominal fluid. In cats, a 2:1 ratio of abdominal fluid creatinine to serum creatinine and 1.9:1 ratio for abdominal fluid potassium to serum potassium is considered diagnostic for uroabdomen.

On cytology, given urine is a chemical irritant, a non-septic neutrophilic inflammatory process is often encountered. Cats will have an increased risk of septic peritonitis if they had bacteriuria from a pre-existing urinary tract infection. It is therefore important to evaluate the slide closely for evidence of bacteria.

### Key point
Uroabdomen can be confirmed if the creatinine of the abdominal effusion is ≥2 times that of the peripheral blood and the potassium of the abdominal effusion is ≥1.9 times that of the peripheral blood.

### Diagnostic imaging
Following initial stabilisation and laboratory evaluation, abdominal imaging can be performed to identify the location of urinary tract injury. The possible imaging modalities include:
- abdominal radiographs;
- abdominal ultrasound;
- computed tomography.

The modality of choice will depend on cost, availability and experience. In veterinary medicine, radiography is often the first choice. Survey radiographs of the abdomen are taken first (Figure 1). These may reveal a generalised loss of serosal detail, suggestive of an abdominal effusion. Occasionally, free gas may also be present within the abdominal cavity. This is more commonly associated with distal urinary tract rupture, at the level of the urethra. The survey radiographs may also reveal the presence of uroliths within the urinary tract (Figure 2). Positive-contrast radiography is subsequently used to identify the exact site of urine leakage. To aid interpretation of the urogenital tract it is advisable to perform an enema to evacuate the colon. A retrourethrocystogram (Figure 3) is useful for highlighting leaks within the lower urinary tract (urethra, urinary bladder) and is often the method of choice in the majority of clinical settings. On the other hand, intravenous urography will allow identification of a leak at any point within the urinary tract.

Ultrasonography allows identification of an abdominal effusion and assessment of the architecture of the kidneys, bladder and other structures. It can be difficult to locate the site of rupture on ultrasound. Identification of a bladder rupture can be improved by instilling an agitated sterile saline solution, containing numerous microbubbles, into the bladder via a urinary catheter (Figure 4).

**How do you stabilise a cat with uroabdomen?**
Surgical exploration and repair of the urinary tract may be the definitive treatment for some cats with uroabdomen. However, before the cat is suitable for surgery, it is...
imperative that it is cardiovascularly stable and the metabolic and electrolyte disturbances have been addressed. The approach to stabilising these patients is summarised in Box 2.

Cardiovascular stabilisation
When the urine enters the abdominal cavity the presence of large osmotically active particles, such as creatinine, draws water from the intracellular and intravascular fluid into
the abdomen. This can result in significant volume depletion and haemodynamic compromise.¹

Intravenous fluid therapy should be started with urgency in any patient presenting with signs of hypovolaemia and perfusion abnormalities. Oxygen supplementation may also be beneficial at this stage. Isotonic crystalloids, such as Hartmann’s or 0.9% saline, have been found to be safe and effective. A balanced electrolyte solution such as Hartmann’s has been considered to improve the acid–base status more rapidly than 0.9% saline.⁷ The typical crystalloid shock rate for cats is 40–60 ml/kg. However, due to the risk of fluid overload, it is now recommended that cats receive crystalloid fluid boluses in increments of one quarter of the total shock volume over 15–20 mins. The clinical status of the cat should be reevaluated before further boluses are administered in order to determine ongoing fluid requirements. Clinical parameters to assess include the patient’s mentation, capillary refill time, heart rate, blood pressure and urinary output.⁷

Electrolyte disturbances
Hyperkalaemia causes decreased cell membrane excitability. Clinically, cats may present with generalised skeletal muscle weakness and/or potentially life-threatening cardiac rhythm disturbances.⁶ It is therefore, advisable to perform an electrocardiogram (ECG) on these patients.⁷ Characteristic ECG abnormalities seen with a mild-to-moderate hyperkalaemia include bradycardia and tall, tented T waves. In the event of severe hyperkalaemia (>8.5 mmol/l) there may be absent P waves, atrial standstill, ventricular tachyarrhythmias and eventually ventricular asystole.⁶,⁷,⁹

Medical management of hyperkalaemia focuses on antagonising the cardiotoxic effects of hyperkalaemia, followed by reducing the serum potassium levels through elimination and redistribution of potassium:⁷

- **elimination**: this is best achieved through the administration of...

---

**Box 2: Summary of the initial stabilisation of uroabdomen**

**Cardiovascular stabilisation:**
- gain intravenous access;
- intravenous fluid therapy;
- provide oxygen as required.

**Hyperkalaemia:**
- **elimination**: promote potassium diuresis with intravenous fluid therapy and actively drain urine from the bladder and peritoneal cavity.
- **redistribution**: shift potassium from the extracellular space to the intracellular space. With dextrose, insulin and dextrose, sodium bicarbonate or β₂-adrenergic agonists.
- **antagonism**: antagonise the cardiotoxic effects of hyperkalaemia with 10% calcium carbonate.

**Metabolic acidosis:**
- intravenous fluid therapy;
- sodium bicarbonate may be considered in severe cases.

**Urine drainage:**
- catheterise the urinary bladder;
- peritoneal fluid drainage;
- peritoneal dialysis may be considered in severely ill patients.

**Pain management:**
- opioids (methadone or buprenorphine);
- adjunctive analgesia (local anaesthesia, gabapentin, ketamine continuous rate infusion).
intravenous fluid therapy with crystalloids. By replenishing the fluid deficit the glomerular filtration rate will improve and increase the excretion of the potassium. For cats with mild to moderate hyperkalaemia (5.5–7.5 mmol/l) fluid therapy, alone is often sufficient. Loop diuretics can also further increase the urinary excretion of potassium; however, they should not be used until the cat is rehydrated, and therefore, are often not indicated in the initial stabilisation.

• **redistribution of potassium:** this involves shifting potassium from the extracellular space to the intracellular space. Options include: a 5–10% dextrose infusion, regular insulin combined with a dextrose infusion, sodium bicarbonate or β2-adrenergic agonists, such as terbutaline. Administration of dextrose will result in the release of endogenous insulin. This will promote the movement of glucose and potassium into the intracellular space. In reality, the exogenous administration of dextrose does not cause a significant rise in endogenous insulin. Therefore, more often, a dose of regular insulin is administered along with a continuous rate infusion (CRI) of dextrose to prevent hypoglycaemia. Sodium bicarbonate or β2-adrenergic agonists are typically only considered if the cat is unresponsive to dextrose and insulin therapy.

• **potassium antagonism:** 10% calcium gluconate can be given intravenously to antagonise the cardiotoxic effect of the potassium on the myocardium. This medication typically works for 30–60 mins. While it does not reduce the potassium levels, it does allow time for the therapy aimed at reducing serum potassium to become effective.

**Metabolic acidosis**
Impaired clearance of hydrogen ions by the kidney leads to metabolic acidosis. Intravenous fluid therapy is the first line therapy for the treatment of metabolic acidosis. Hartmann’s may rectify the imbalance faster than 0.9% saline as the lactate is metabolised to bicarbonate which acts as an alkalinising agent. If the cat is severely acidotic, pH <7 or HCO3- is <12 and the acid–base status is not improving with fluid therapy, then sodium bicarbonate may be considered. It is important to give small, incremental doses, rechecking the blood gas values after each dose to avoid over supplementation and subsequent alkalosis.

**Urine drainage**
A urinary catheter should be placed into the bladder. This enables the urine output to be monitored more accurately. Regular drainage of the bladder will also keep the bladder decompressed. This helps to reduce the hydrostatic pressure within the bladder, subsequently reducing urine leakage into the abdomen. It will also allow small tears within the bladder time to heal without surgical intervention.

Drainage of the urine residing within the peritoneal cavity using a needle or peritoneal catheter has also been...
State of the art

**Pain management**

Analgesia should not be overlooked as the chemical peritonitis caused by urine in the abdominal cavity can be very painful. Opioids such as methadone and buprenorphine can be used. Occasionally, a multimodal analgesic approach is required. In addition to the opioids, a ketamine CRI, gabapentin or local anaesthesia can be considered. It is important to be aware that ketamine is excreted unchanged in the urine. Therefore, until the uroabdomen is drained ketamine can be continuously reabsorbed, resulting in prolonged sedation and anaesthesia.7

In the presence of hypovolaemia and azotaemia, the use of non-steroidal anti-inflammatories are not recommended. In addition, α2-adrenergic agonists (such as dexmedetomidine) should be avoided. The bradycardia, peripheral vasoconstriction and decreased cardiac output seen with α2-adrenergic agonists may exacerbate the cardiotoxic effects of the hyperkalaemia.

found to improve patient stabilisation. The catheter or needle can be aseptically placed in a conscious cat using a local anaesthetic block.1,2,5,7 Peritoneal dialysis may be considered in cats with severe azotaemia, hyperkalaemia and acid-base derangements that are not responsive to the intravenous fluid diuresis and pharmacological intervention mentioned.5,24

**What is the prognosis?**

The prognosis for cats with uroabdomen depends on a number of factors. These include: the presence of concurrent conditions such as musculoskeletal injury, cause and location of the rupture, severity of metabolic disturbances and the management of these cases.2,3,5 The prognosis is improved for cats sustaining iatrogenic injuries, those without concurrent conditions or injuries, and cats receiving early diagnosis with rapid management and stabilisation.5,7,15

**References**

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Urinalysis in cats 2: measurement of USG and what the results mean

Measurement of urine specific gravity (USG) is a useful test of renal function and should be performed during routine urinalysis. USG reflects the total solute concentration of the urine. USG measurement allows classification of hypersthenuria, isosthenuria and hyposthenuria. The majority of healthy cats will have a USG >1.035. A lower USG may be consistent with renal disease or other conditions. Urine dipsticks are unreliable for assessment of USG and if urine is turbid, USG should be measured on the supernatant after centrifugation.

Measurement of urine specific gravity (USG) is the only test of renal function (ie, the kidneys’ ability to modify the solute content of the urine via dilution and concentration) performed during routine urinalysis.

USG reflects the total solute concentration in the urine. Osmolality is more accurate than USG as it depends solely on the number of particles in the solution (not a combination of number, particle size and molecular weight as in USG). However, osmolality is not readily measured in practice, while USG is easy and can be performed in-house using a refractometer.

Care must be taken to obtain accurate, repeatable and reliable USG readings from a digital or analogue refractometer. USG measurements should be made prior to the commencement of any treatment that has the potential to alter USG (Table 1).

\[
USG = \frac{\text{density (weight) of urine}}{\text{density (weight) of distilled water (}= 1.000)}
\]

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This is the second in a five-part series on urinalysis. See Urinalysis in cats 1: urine collection, storage and initial assessment. Feline Focus 2017: 3(2): 41–50. Part 3 will cover urine dipstick tests. Parts 4 and 5 will look at microscopic analysis of urine.
**How to...**

**Measurement of USG**

A refractometer (Figure 1) provides an indirect measure of specific gravity (and osmolality) by detecting light wavelength absorption (refraction of light) by solutes in urine relative to light refraction in air — the so-called refractive index (RI).

The RI of urine is dependent on the solute concentration, chemical composition of the solute and temperature of the urine sample.

Refractometers only measure the RI of the soluble solids in fluid (ie, electrolytes, protein, glucose, urea and creatinine). Suspended particles in urine (eg, cells, casts and most...

**Table 1: Checklist to help ensure accurate results when using a refractometer to measure urine specific gravity (USG)**

<table>
<thead>
<tr>
<th>Check points</th>
<th>Action/considerations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ensure regular refractometer calibration</td>
<td>Ideally, refractometer calibration should be checked daily with distilled water (USG 1.000) or a 5% NaCl solution in distilled water (USG 1.022). Do not use commercially prepared protein standards for instrument calibration as they contain non-protein preservatives that increase refractive index values.</td>
</tr>
<tr>
<td>Use temperature-compensated refractometers</td>
<td>Temperature affects the density of urine and hence the USG. For non-temperature compensated refractometers, read at or near 20°C for accurate measurement.</td>
</tr>
<tr>
<td>Avoid USG measurements on turbid urine</td>
<td>USG should be determined on the supernatant after centrifugation of turbid urine.</td>
</tr>
<tr>
<td>Reading USG results &gt;1.035 off refractometer scales that only read up to 1.035</td>
<td>Dilute urine 1:1 with distilled water and then double the last two digits of the reading obtained to give the true USG.</td>
</tr>
</tbody>
</table>
| Factors increasing USG                     | Consider:  
  • glucosuria — a concentration of 1 g/dl (55.5 mmol/l) of glucose in urine will increase the USG by 0.004–0.005 units  
  • proteinuria — a concentration of 1 g/dl (10 g/l) of protein in urine will increase the USG by 0.003–0.005 units;  
  • turbidity of urine;  
  • cold temperatures;  
  • free haemoglobin;  
  • increased lipid content;  
  • radiographic contrast media                                                                                                                                                                                                                                                                                                                                                                                                   |
| Factors decreasing USG                    | Measure USG prior to fluid therapy and diuretics                                                                                                                                                                                                                                                                                                                                                                                                                                                        |
| Take appropriate care of the refractometer | Rinse and dry the clear optical measuring surface and the underside of the cover plate that comes into contact with the measuring surface. Avoid scratching the clear optical measuring surface.                                                                                                                                                                                                                                                                                                |

**Key point**

Particles in urine can interfere with USG measurement, hence, turbid urine should be centrifuged and the USG determined on the supernatant.
crystals) do not refract light and consequently do not affect the RI of urine. Suspended particles in urine do, however, interfere with light transmission (reducing the clarity of the urine), making the demarcation line on the refractometer’s reticle more difficult to read (Figure 2). Consequently, if the urine is turbid, the USG should be determined on the supernatant after centrifugation.

Traditionally, it has been advised that veterinary refractometers with specific feline USG scales be used as it was reported in 1956 that feline urine had a higher relative specific refractivity than human or canine urine; a conversion factor of $0.846 \times \text{medical refractometer USG} + 0.154$ was suggested. However, recent studies suggest that feline urine is not different from other species urine and that the issue with the original paper was one of concentration rather than species. Some ‘feline-specific’ refractometers have been demonstrated to be inaccurate in clinically relevant ranges and medical/canine refractometers have now been recommended.

Digital refractometers are now available and one study compared the use of hand-held optical analogue refractometers with cat-specific digital refractometers. USG measurements from both instruments were highly correlated with urine osmolality, suggesting that both devices were valid in assessing feline USG in clinical practice. The digital
How to...

**Dipsticks and USG measurement**

Dipstick methods are available for approximating USG but are not recommended for cat urinalysis, as USG values are falsely decreased when the urine pH is >6.5 or glycosuria is present. Furthermore, as maximal dipstick USG readings are 1.025–1.030, USG dipsticks are unsatisfactory for the detection of adequate renal concentrating ability in cats.²⁻⁴,⁶⁻⁹

**Interpretation of USG measurements**

USG in healthy adult cats may range from 1.001–1.085, although a recent large-scale study in 1040 apparently healthy adult cats reported that 88% of the cats concentrated their urine to a USG of >1.035 on a random single urinalysis.⁸ The same study also revealed that dietary modification had only a modest effect on USG, with increasing dietary moisture content more likely to lower USG in female cats.⁸

USG must always be assessed and interpreted in conjunction with patient health, hydration status and azotaemia:³⁻⁴

- hyposthenuria (USG <1.008): implies that the kidneys are capable of diluting the glomerular filtrate. While persistent hyposthenuria usually suggests that renal dysfunction is not present, some cats with renal dysfunction are still capable of producing hyposthenuric urine.³ Urinary tract infections (UTIs) can also result in hyposthenuria.

- isosthenuria (USG 1.008–1.012): indicates that the glomerular filtrate has not been altered by the kidneys.¹² Isosthenuria occurs occasionally in healthy cats but, when accompanied by dehydration and azotaemia, is indicative of decreased renal function. Persistent isosthenuria in a non-azotaemic cat is suggestive of a concentrating defect.

- hypersthenuria (USG >1.012): demonstrates that the kidneys are capable of concentrating the glomerular filtrate to some extent. Cats should be able to concentrate to at least 1.040 in the face of dehydration or azotaemia. Some cats with less than 25% functional renal mass have been able to concentrate to >1.040, suggesting that 1.040 may be a conservative cut-off for differentiating pre-renal from renal azotaemia. It has been proposed that, in azotaemic cats, >1.045 implies adequate concentration, 1.040–1.045 is questionable and <1.040 inappropriate.³⁻⁹ The production of very concentrated urine (>1.050) in a dehydrated or azotaemic cat suggests reduced renal perfusion (hypovolaemia, hypotension or cardiac disease) rather than renal disease.⁹

**Key point**

Measurements of USG should be made prior to any treatment as this may alter the results.
Difficulties in interpretation in renal dysfunction
Difficulties in interpreting feline USG may be encountered with cats in renal dysfunction that retain concentrating ability for a variable period after the onset of azotaemia. Cats with 58–83% reduction in renal mass can still produce hypersthenuric urine, with a USG of 1.022–1.067. Consequently, some of these cats could be misclassified according to the USG as having pre-renal azotaemia.3,9,10

Neonates
Assessment of USG in neonates is also challenging. In comparison with adults, neonates tend to have poorer renal concentrating ability. The USG in random samples from healthy neonatal kittens is typically low (1.006–1.007). USG readings of up to 1.038 have been observed in kittens by 4 weeks of age and up to 1.080 may be possible by 8 weeks of age.3,11

USG and proteinuria tests
Routine evaluation of USG is required for interpretation of some of the test results from a complete urinalysis. The amount of any substance in the urine, such as protein, must be interpreted in the light of the USG. For example, a 2+ dipstick protein result on a urine sample with a USG of 1.060 indicates mild proteinuria, in contrast to a 2+ dipstick protein result on a urine sample with a USG of 1.007, which indicates a significant proteinuria.4

References

This article has been adapted from: George Reppas and Susan F Foster. Practical urinalysis in the cat 1: urine macroscopic examination ‘tips and traps’. J Feline Med Surg 2016; 18: 190–202.

Key point
USG must always be assessed and interpreted in conjunction with patient health, hydration status and azotaemia.
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‘High-rise’ syndrome: cats don’t always land on their feet

‘High-rise’ syndrome is a term used to describe a group of injuries sustained when cats fall from a height. Common injuries include thoracic injuries, limb fractures, spinal and orofacial injuries and other soft tissue trauma such as ruptured bladder. Cases should be triaged with attention to underlying life-threatening conditions such as pneumothorax and pulmonary contusions. Advice should be given to owners to prevent high-rise syndrome, such as using meshes and nets to allow windows to be opened and balconies safely accessed.

The term ‘high-rise’ syndrome is used to describe the injuries suffered by cats falling from a height, for example from a balcony or window, often when cats are kept in flats or apartments. The term has been used for many years following early publications looking at the injuries suffered in such cases.1,2 Cats falling from heights (usually second floor or higher) suffer a range of injuries (Figure 1), many very severe, and this article discusses the types of injuries sustained, which is helpful when assessing an emergency trauma case with this type of history.

Samantha Taylor graduated as a veterinary surgeon in 2002 from the Royal Veterinary College, UK, and spent time in first and second opinion practice before becoming the International Cat Care Resident in Internal Medicine in 2006. She became a European Specialist in Internal Medicine in 2009 and a Royal College of Veterinary Surgeons Veterinary Recognised Specialist in Feline Medicine in 2010. She is currently the Nurse and Technician Distance Education Coordinator at International Cat Care and editor of Feline Focus.

Key point

Cats with high-rise syndrome suffer various orthopaedic and soft tissue injuries, some more life-threatening than others and increasing in severity with the height of the fall.

Figure 1: High-rise syndrome consists of multiple injuries and these cases (like this cat with head and musculoskeletal injuries) can be hard to manage with long recovery periods. (Courtesy of Laura Owen)
Injuries caused by falling

Various different traumatic injuries are described following falls, both orthopaedic and soft-tissue. As would be presumed, the higher the fall, the more severe the injuries sustained, although this is true up to the seventh story, after which interestingly, studies (including only small numbers of cats) show minimal fatalities, with one cat falling from the 32nd floor and only suffering a mild pneumothorax and a chipped tooth. Injuries sustained may be influenced by fall height due to the cat’s ability to change its body position and land on its feet, this ability resulting in limb fractures from shorter falls, but on longer descents its vestibular system may be less able to adjust body position, meaning the cat lands with legs horizontally, sustaining soft tissue injuries. Additional factors, such as the substrate on which the cat lands and obstructions to the fall, will influence injury.

The most common injuries tend to be thoracic trauma, limb fractures and orofacial injuries:

- **thoracic injuries:** (present in 34–90% of cases) pneumothorax and pulmonary contusions are the most common. The prevalence of thoracic injury in studies of cats with high-rise syndrome varies, perhaps due to differences in criteria to assess injury, and selection of patients to radiograph. The high prevalence in some studies suggests routine radiography of cats after falls should be advised.

- **limb fractures:** reported in around 50% of cases and may affect the distal limb more frequently in younger cats, in the region of the growth plates.

- **spinal injuries:** some studies show a high frequency of spinal injuries, and interestingly in this study the falls were from lower heights than in other studies, perhaps leaving cats no time to adjust their body position and land on their limbs.

- **carpal joint injuries:** these injuries are generally rare in cats, but in one study 72.6% cats with such injuries had fallen from a height. This study also noted that carpometacarpal joint injuries in particular were associated with falling from the third floor or lower, whereas antebrachiocarpal joint injuries were seen in cats falling from the fourth floor or higher, illustrating that the height of the fall can affect the type of injury sustained.

- **orofacial injuries:** generally, when falling, the limbs and body impact before the head. However, significant orofacial trauma can occur, including hard palate fracture (Figure 2), mandibular fracture, mandibular symphyseal separation, tongue and soft tissue injury and dental trauma. Epistaxis is often observed, and
Immediate management of the cat with a history of falling

If a cat is presented after a fall, it should be triaged as with any other trauma (eg, a road traffic accident), focusing on the following:

- **respiratory tract**: pneumothorax and pulmonary contusions are common, so oxygen should be provided and close observation of respiratory pattern. Cats with pneumothorax will have a rapid and shallow breathing pattern, absent breath sounds and increased resonance on percussion. Thoracic radiography may be indicated, but heavy restraint must be avoided in all dyspnoeic cats.

- **cardiovascular system**: pulse rate, strength and rhythm should be recorded, along with capillary refill time and mucous membrane colour.

- **spinal injuries**: cats should be handled extremely carefully if spinal injury is suspected, with movement to other areas (eg, radiography) minimised. Mentation should also be noted, and a full neurological examination planned once the cat is stable.

- **open fractures**: such fractures should be assessed by the veterinary surgeon as soon as possible, as early treatment may prevent significant bacterial infection.

- **intravenous access**: if possible (depending on respiratory compromise) intravenous access should be obtained rapidly, to allow administration of medications and fluid therapy.

- **analgesia**: cats with high-rise syndrome will be anxious and in pain. Pain scoring systems can be useful to monitor the effect of the analgesia. Opioids are a good choice due to minimal cardiovascular effects.

- **anxiety and fear**: a cat that has suffered recent trauma may be incredibly fearful; handle the cat kindly and quietly and keep away from dogs and other sources of fear. Keep to Cat Friendly Clinic principles and provide a soft bed and kind and gentle attention while the cat is being treated.

- **further assessment of injuries**: diagnostic imaging may be indicated to assess for abdominal injury (eg, ruptured bladder) and limb fractures, but life-threatening conditions should be managed first.

Key point
Abdominal trauma can cause traumatic pancreatitis which is very challenging to treat.

Predispositions to high-rise syndrome
Most studies show cats affected by this syndrome are young, many less than 1 year of age. This is presumably due to inexperience, curiosity and more exuberant play. It occur. Traumatic pancreatitis, or pancreatic duct rupture are reported after falls, and the outcomes were not favourable. This type of pancreatitis is uncommon but very challenging to treat.
Keeping cats safe

**Prevention of high-rise syndrome**

High-rise syndrome is completely avoidable by taking steps to deny cats access to areas from which they can fall. While access to a balcony, or fresh air from a window may be very pleasant to a cat kept indoors (Figure 3), especially on a warm day, there are ways to prevent high-rise syndrome and avoid a stuffy house:

- **Furniture**: Move furniture cats could use to climb to access higher level windows.
- **Windows**: Open windows can be made safe with limiters that allow the window to only open a certain amount (designed to keep children safe), but remember cats, and particularly kittens, can squeeze through very small spaces, or get stuck trying.
- **Meshes**: Meshes or barriers that cover the open windows are also available and may be better, allowing the window to fully open and remain safe (look in the child and pet safety sections); some are designed specifically for this purpose.
- **Balcony Nets**: Balcony safety nets and meshes are available, just make sure all escape routes are covered. Mesh may prevent climbing over the rails, but many cats can squeeze through fencing and barriers designed for keeping people safe.

**Tip**

Young cats are more likely to fall than older cats. Advise new kitten owners to use mesh and nets over windows and balconies.

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is reported that cats fall when playing with other cats, balancing along window and balcony ledges, and when chasing birds or butterflies. Clearly, cats living in urban areas and those kept completely indoors are more likely to fall than cats with free access outdoors in rural environments. In certain parts of the world cats are commonly kept indoors in high-rise buildings and flats, and in warmer climates balconies and open windows are more common. There is a peak in the incidence of high-rise syndrome in warmer months, when there is opportunity to reach open windows and doors to balconies.

**Conclusions**

High-rise syndrome can be very serious, and fatal in some cases. Multiple different injuries may be sustained, some more life-threatening than others. Cases should be assessed with the knowledge of common injuries caused by falls, and life-threatening conditions such as a pneumothorax managed before other injuries such as long bone or jaw fractures. Clients should be advised how to prevent high-rise syndrome by securing windows and balconies, and considering where mesh and nets can be used to prevent cats falling. Nurses and technicians in areas where cats are often kept indoors in high-rise flats should discuss safety with cat and kitten owners visiting their clinics to prevent accidents.

**References**

2. Whitney WO and Mehlhaff CJ. High-

For more details on the International Cat Care keeping cats safe campaign go to:
icatcare.org/about-us/our-campaigns/keeping-cats-safe
Lethal lilies

Eating any part of the lily – flowers, leaves, stem or pollen – is EXTREMELY DANGEROUS TO CATS and can cause kidney damage and even death.

IF YOU THINK YOUR CAT HAS Eaten ANY PART OF A LILY, CONTACT YOUR VET IMMEDIATELY.

Lilies (Lilium species) and day lilies (Hemerocallis species) are highly toxic to cats.

Download free practice posters at http://icatcare.org/advice/keeping-cats-safe

icatcare.org/keeping-cats-safe

INTERNATIONAL CAT CARE – KEEPING CATS SAFE CAMPAIGN

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A 2.5-year-old, male neutered, domestic shorthair cat presented with a 2-day history of lethargy, anorexia and vomiting and a more chronic history of polydipsia. A diagnosis of diabetic ketoacidosis was made by documenting elevated blood glucose and ketones. The cat was also diagnosed with pancreatitis. Treatment involved fluid and insulin therapy, as well as management of nausea, pain and attention to nutrition. Nursing priorities included accurate fluid therapy, close monitoring of blood glucose, monitoring for pain and encouraging the cat to eat using techniques to reduce stress.

This case report describes the initial treatment of feline diabetic ketoacidosis with concurrent pancreatitis and discusses the nursing considerations that this patient required until discharge. Diabetes mellitus (DM) is a common endocrine disease seen in general practice and owners report clinical signs such as polydipsia, polyuria and weight loss. The cats with DM are often bright, well-hydrated and frequently, therefore, can be managed as day patients. This case report describes the treatment and nursing considerations for a cat with diabetic ketoacidosis and pancreatitis.

History
Pickles, a 2.5-year-old, male neutered, domestic shorthair, presented as a routine appointment with the owner reporting a 2-day history of lethargy, anorexia and two observed episodes of vomiting. A more detailed history revealed that the owners thought he may have been drinking more for the preceding 2 weeks. Clinical examination revealed:

- generalised weakness with depressed mentation;
- a skin tent suggesting a level of dehydration of approximately 8%;
- mucous membrane colour was pink with a capillary refill time of less than 2 s;
- abdominal palpation was unremarkable;
- thoracic auscultation was unremarkable. Heart rate was approximately 120 bpm, regular rate with no pulse deficits;
- rectal temperature was low-normal at 37.5°C; and
- body weight was 4.6 kg (it had been 5 kg at last vaccination 2 months previously).

Following the consultation Pickles was admitted for blood sampling.
Case study

(in-house serum biochemistry with electrolytes and packed cell volume) and intravenous fluid therapy.

Laboratory results
Salient serum biochemistry results are shown in Table 1.

Table 1: Biochemistry results

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Patient results</th>
<th>Reference interval</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alanine aminotransferase</td>
<td>506</td>
<td>20–100 IU/l</td>
</tr>
<tr>
<td>Total bilirubin</td>
<td>17</td>
<td>2–10 μmol/l</td>
</tr>
<tr>
<td>Phosphate</td>
<td>0.95</td>
<td>1.10–2.74 mmol/l</td>
</tr>
<tr>
<td>Glucose</td>
<td>2.76</td>
<td>3.9–8.3 mmol/l</td>
</tr>
<tr>
<td>Sodium</td>
<td>131</td>
<td>142–164 mmol/l</td>
</tr>
<tr>
<td>Potassium</td>
<td>2.3</td>
<td>3.7–5.8 mmol/l</td>
</tr>
</tbody>
</table>

- Fructosamine was high at 570 μmol/l (reference interval 190–350), consistent with prolonged hyperglycaemia and confirming diabetes mellitus.
- Serum feline pancreatic immunoreactivity was measured and was severely elevated at 53.0 μg/l (reference interval 2.0–7.0).
- Serum ketones were crudely measured in the practice by placing a drop of serum onto the ketone reagent pad on a urine dipstick – the results were positive (+++).

Diagnosis
Pickles was diagnosed with diabetic ketoacidosis (DKA) with concurrent pancreatitis. Note that, ideally, blood pH is measured to make this diagnosis but the facilities to do this were not available.

Nursing considerations
- Correction of hyperglycaemia and acid-base/electrolyte abnormalities.
- Monitoring blood glucose following commencement of insulin treatment.
- Correction of dehydration.
- Provision of analgesia, anti-nausea/antiemetic and gastroprotective medications for pancreatitis.
- Attention to nutrition.
- Care of the hospitalised cat to ensure minimal stress, including providing a warm bed as the cat's...
temperature is low-normal.

- Demonstration to, and education of, the owner in home management of the newly diagnosed patient with diabetes with insulin.

Treatment

Intravenous fluid therapy

The ‘textbook’ case of DKA usually involves consideration of several commonly seen metabolic derangements and in this case the following need managing:

- hyperglycaemia;
- hyponatraemia;
- hypokalaemia;
- hypophosphataemia; and
- metabolic acidosis.

The two primary choices for fluid therapy in DKA cases are 0.9% sodium chloride solution and lactated Ringer’s solution (LRS; Hartmann’s). Although 0.9% sodium chloride solution is often selected, it is unbuffered and acidifying hence can theoretically make acidosis worse. The addition of potassium is suggested whether using either solution as neither contains the requisite concentration of potassium. LRS does contain some potassium in the form of potassium chloride (5 mmol/l), but additional potassium is required as considerable shift in serum potassium into cells can take place when treatment commences, worsening hypokalaemia.

LRS with additional supplementary potassium chloride (30 mmol in 500 ml) was selected to provide intravenous fluid therapy (Figure 1).

The amount of fluid to administer should be accurately calculated rather than using an arbitrary rate. In this case the cat was 8% dehydrated indicating a deficit of 8 x 4.6 x 10 = 368 ml, to be replaced over 24 h = 15 ml/h. Maintenance requirements should be added (50 ml/kg/day = 10 ml/h) as well as ongoing losses (Pickles was monitored for any further vomiting) giving a rate of 25 ml/h with close monitoring of vital parameters (heart rate, respiratory rate) and regular thoracic auscultation to detect fluid overload.

Key point

Fluids should be supplemented with potassium when treating a cat with DKA as they can become severely hypokalaemic when treated, as potassium shifts into cells.

Tip

Fluid administration rate should be calculated accurately based on the level of dehydration and in response to closely monitored fluids, rather than using an arbitrary fluid rate in units of maintenance.
Case study

Insulin therapy
After 4 h of fluid therapy (which will lower blood glucose and ketones) lente insulin was administered at a dose of 0.25 IU/kg subcutaneously every 12 h, as unfortunately there was no neutral insulin available. Alternatives to intermittent injections of insulin include infusions of insulin.

Key point
Soluble/neutral insulin can be given to cats with DKA as intermittent injections or as a continuous rate infusion. Protocols using glargine insulin are also available.

Analgesia, antiemetic and gastroprotective medication
Analgesia was provided with buprenorphine at 0.015 mg/kg intravenously (IV) q8–12h for the first 24 h of hospitalisation before tapering to 0.01 mg/kg IV q12h for following 24-h period.

Antiemetic treatment was provided with maropitant 1 mg/kg subcutaneously administered on the first and second day of hospitalisation. It was discontinued after the second dose as Pickles was eating well voluntarily with no further episodes of vomiting following admission.

Gastroprotectant medication was given using ranitidine 2.5 mg/kg IV (administered slowly) q12h throughout the period of hospitalisation to help treat any gastrointestinal irritation indicated by the vomiting. This medication was discontinued at discharge.

Nutrition
As nutrition is important for recovery, voluntary intake was encouraged; daily calorific requirements were calculated with a plan to place a naso-oesophageal feeding tube if needed. A low carbohydrate diet is suggested in feline patients with diabetes mellitus, but initially tempting and familiar foods is indicated as it is not appropriate to offer a diet ideal for longer term at a time when a cat is stressed or nauseous, as this could result in aversion to that food. Pickles ate well within 12 h of admission following supportive care and medications (Figure 2).

Monitoring the cat with DKA
In clinical situations where repeated blood glucose measurement is required, collection from the marginal ear vein is suggested (Figure 3). This technique allows collection of the very small volume of blood required for portable glucometers without unnecessary handling of patients for jugular or cephalic sampling. In this case the following monitoring was performed:
• hourly blood glucose for the first 12 h after insulin injection;
• measurement of electrolytes after 1 and 4 h of treatment;
• initially, a 30 min check of pulse, respiratory rate and thoracic auscultation for fluid overload, reduced to hourly after initial positive response then 4 hourly once stable;
• regular assessment of demeanour for signs of nausea, pain or distress;
• free catch urine samples were collected to measure urine ketones.

Outcome
Pickles responded well to treatment. Blood glucose fell into the normal range following insulin administration and electrolytes normalised after 4 h of treatment. Fluid therapy was reduced and discontinued once hydration was normal and Pickles was eating.

Key point
Newly diagnosed diabetics need close monitoring as their insulin requirement may fall as hyperglycaemia and any complicating conditions (eg, pancreatitis) resolve.

Pickles was discharged on day 4 with adequate voluntary food intake. Analgesia had been discontinued the day before without signs of Pickles’ condition worsening. He was discharged on 1 IU lente insulin twice daily with a low carbohydrate diet for the owner to slowly introduce at home.

He was re-examined a week later and a glucose curve performed showing good control, he was doing clinically very well. He was scheduled to be re-assessed again in another week.

Discussion
The four diagnostic criteria for DKA are as follows;
• hyperglycaemia,
• glucosuria,
• ketonaemia/ketonuria; and
• metabolic acidosis.

Only the first three of these criteria were met in this case with the fourth being assumed as blood gas analysis was not available. The diagnosis of pancreatitis was initially made using a bench-side test and confirmed in the laboratory. Pancreatitis is particularly common in cats with diabetes and may be underdiagnosed.3,4

A case such as this illustrates the numerous nursing aspects that need to be considered in the hospitalised cat including management of fluid therapy, monitoring and attention to nutrition, analgesia and antiemetic therapy. Most cats that present with DKA have a concurrent disease or underlying cause that precipitates the shift from diabetes mellitus to DKA. Hepatobiliary conditions such as pancreatitis, hepatic lipidosis or cholangiohepatitis should be considered in cats with DKA.

The importance of intravenous fluid
therapy is highlighted both to correct the dehydration as well as treat the electrolyte/acid base derangement. Fluid therapy should be accurately calculated and the patient monitored closely. The supplementation of potassium in this case was of the utmost importance. Patients that present with DKA often have whole body potassium deficits. Correction of the acidosis along with the administration of insulin can worsen the hypokalaemia as the potassium moves intracellularly. Some cases with severe hypophosphataemia require supplementation, but in this case the hypophosphataemia was mild and resolved rapidly.

Analgesia is an integral part of the management of feline pancreatitis, and pain should be assumed as it can be difficult to evaluate in the cat. Opioid analgesia is most commonly selected, with buprenorphine and methadone used most often, with non-steroidal anti-inflammatories often being avoided in hypovolaemic and dehydrated patients. The effective dose, route and frequency of the chosen opioid often requires frequent reassessment using pain scoring schemes such as the Colorado State University Feline Acute Pain Scale or Glasgow Composite Pain Scoring Scheme.

References