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Emergency Patients - An Organ Approach Mini Series

Session Three: Nursing Care of the Renal and Urinary Emergency Patient

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NURSING CARE OF THE RENAL AND URINARY EMERGENCY PATIENT

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NURSING CARE OF THE ACUTE RENAL PATIENT

The kidney's function is to eliminate metabolic waste materials and help maintain homeostasis by manipulating the composition of blood plasma. The kidneys achieve homeostasis by regulating acid-base, fluid and electrolyte balance and producing hormones (erythropoietin, renin, and calcitriol). In the normal animal, sodium, potassium, chloride and nitrogenous waste must be maintained within narrow concentration limits. The acute failure of the kidneys to perform their function was previously known as acute renal failure. We now recognize that an acute disruption of kidney function correlates with injury but may not progress to permanent kidney failure. Thus, 'acute renal failure' is now termed acute kidney injury (AKI). Acute kidney injury is characterized by the rapid loss of nephron function, resulting in azotemia and fluid, electrolyte, or acid-base abnormalities. The decrease in kidney function that occurs with AKI is multifactorial and includes cellular damage and decreased intrarenal blood flow. This discussion will focus on the nursing management of AKI.

Physiology

The functional unit of the kidney is the nephron. It is made up of a glomerulus, proximal tubule, loop of Henle, distal tubule, and collecting duct. Up to 25% of the cardiac output goes to the kidneys. Blood enters the glomerulus; the hydrostatic pressure in the glomerular capillaries forces some plasma into Bowman's capsule. This fluid is called glomerular filtrate; the glomerular filtrate moves into the proximal convoluted tubules and becomes tubular filtrate. As the tubular filtrate passes through the tubules of the nephron, some of the constituents (sodium, potassium, chloride, calcium, magnesium, glucose, amino acids, bicarbonate and water) are reabsorbed and other constituents are secreted (hydrogen, potassium and ammonia) from the peritubular capillaries into the tubular filtrate. Once the tubular filtrate reaches the renal pelvis it is now known as urine and moves into the bladder via the ureters.

Pathophysiology

Acute kidney injury is caused by a number of conditions or events. Some insult initiates a cascade that leads to renal hypoperfusion and hypoxia causing tubular epithelia damage and death. In addition, there may be a reduction in glomerular capillary hydrostatic pressure and glomerular filtration rate; this reduction leads to decreased urine production and impairs regulation of water and solute elimination.

Acute kidney injury may be caused by decreased blood flow to the kidneys (prerenal), disorders that disrupt structures in the kidneys (intrinsic renal) or disorders that interfere with elimination of urine from the kidneys (post renal).

Common causes of AKI

Prerenal

- Hypovolaemia
- Dehydration
- Hypotension

Intrinsic renal

- Toxins
- Ethylene glycol
- Grapes/raisins

- Lillies
- Evenomation
- Drugs
- Cisplatin
- NSAIDs
- Aminoglycoside
- Infectious
- Leptospirosis
- Pyelonephritis

Postrenal

- Renal/urethral calculi
- Prostatic disease
- Ureteral, bladder, urethral perforation
- FLUTD leading to urethral obstruction

Clinical Presentation & Assessment

History

The patient may report a history of sudden onset of anorexia, vomiting, diarrhea, changes in urination and listlessness. Additional complaints may include seizures, ataxia, or halitosis. Owners should be thoroughly questioned about potential exposure to toxins, ingestion of medications and recent medical procedures (including human preparations).

Clinical Examination

Abnormalities on clinical examination may include oral ulcerations including the tongue, decreased skin elasticity and tacky mucous membranes, tachypnoea, bradycardia and hypothermia. Abdominal palpation may reveal large, painful and/ or swollen kidneys. Patients may have a large non-expressible bladder, suggestive of a urethral obstruction. Patients with AKI generally do not display signs of chronic illness (muscle wasting, weight loss, and poor hair coat). Patients previously treated with fluids may show signs suggestive of fluid overload (generalized oedema, chemosis, and crackles on thoracic auscultation).

Laboratory Evaluation

Blood should be taken for a complete blood count (CBC), biochemistry profile, electrolytes, and blood gases. In addition, a urine sample should be obtained for analysis and possible culture.

A CBC may reveal anaemia, although AKI patients do not usually present with anaemia. Changes may be seen in haematocrit and total protein, suggesting dehydration (decreased PCV and TP). An increase in white cell count may suggest infection or inflammation.

Increases in serum creatinine, urea nitrogen, phosphorus, and potassium are usually consistent with decreased urine output. Calcium may vary.

With the kidneys having a major role in electrolyte and acid base regulation both parameters should be assessed. Hyperkalemia is the most common electrolyte abnormality. Moderate to severe metabolic acidosis may be present.

A urine specific gravity of 1.008–1.012 represents the inability of the kidneys to dilute or concentrate urine. A concentrated urine suggests that the azotaemia is prerenal. Active urine sediment (cast, red and white cells, bacteria and crystals) is seen in acute kidney injury. Calcium oxalate crystals suggest ethylene glycol toxicity.

In hospital, tests are available for testing the presence of ethylene glycol toxicity.

Imaging

Survey radiographs may reveal the size and shape of the kidneys. In chronic renal failure the kidneys may be small and irregular. In AKI, the kidneys may be normal or enlarged. A distended bladder, abdominal or retroperitoneal fluid in the case of a perforation, Calculi and prostatomegaly may be evident.

Ultrasonography can help to evaluate renal architecture, for example bright (hyperechoic) renal cortices may be seen in ethylene glycol toxicity, and also possible ureteral obstruction.

Other

Renal biopsy can be diagnostic and prognostic for intrinsic renal failure.

Treatment

When possible, treatment of the underlying cause should be undertaken. This may mean discontinuation of a drug or drugs, correcting hypoperfusion or administering specific antidotes for toxins. In general therapy includes correction of fluid imbalances, correction of electrolyte and acid base disorders, promotion of diuresis, supportive care and nutritional support.

Catheter Placement

A peripheral catheter is placed initially but a multilumen central catheter is advantageous. A multilumen catheter will allow for the simultaneous administration of fluids, drugs, the collection of blood samples and / or central venous pressure (CVP) measurements. Drugs such as mannitol, with osmolalities greater than 700 mOsm, cannot be administered peripherally without causing severe irritation to the vessel.

A urinary catheter will greatly facilitate care of the patient. It will allow the quantitation of urine, which helps in addressing fluid needs. The catheter should be placed using aseptic technique along with a closed urinary collection system; this will help to minimize urinary catheter related infection.

Correction of Fluid Imbalances

The goal is to induce diuresis so as to decrease serum urea nitrogen and creatinine as well as potassium. If the patient is showing signs consistent with hypovolemia then fluid deficits will need to be corrected immediately. This usually entails the administration of a crystalloid at up to "shock doses" of 50–55 ml/kg in the cat, this is commonly administered as a 15-20ml/kg bolus, or ‘fluid challenge’ and then the patient’s clinical response reassessed before subsequent bolus’ are administered. If the patient's total protein or albumin is 3.0 mg/dl or 1.5 mg/dl respectively then synthetic colloids might be used. Blood products are considered if the PCV is less than 20%. If the patient is simply dehydrated then the product of the percentage dehydration and the body weight equals the volume of fluids necessary to correct dehydration. In addition, maintenance (normal losses) fluids at a dose of 50–75 ml/kg/ day are added to the fluid therapy plan. Finally, abnormal losses (vomiting, diarrhea, third space and excessive urination) are included in the plan and replaced ml for ml. The total volume is replaced over 6–12 hours. Care must be taken not to fluid overload the patient. The primary reason for fluid overload is failure to adjust the fluid administration rate in the face of decreased urine production. The oliguric or anuric patient is incapable of effectively excreting an excessive fluid load. Body weight and indices of hydration should be monitored closely. An early sign of fluid overload is an increasing respiratory rate and effort. Crackles may be auscultated. Chemosis and subcutaneous edema are late signs. CVPs greater than 10 cm H_2O or a significant increase in a short period of time may be indicative of fluid overload.

Correction of Electrolyte and Acid Base Disorders

Life-threatening hyperkalemia and severe metabolic acidosis are common complications in AKI. There are various options for treating hyperkalemia and are dependent on the severity of the elevated potassium level. This includes promoting excretion or dilution with fluid therapy; administration of sodium bicarbonate to correct acidosis and promote H^+ and K^+ ion exchange across cells; administering glucose and insulin to promote the movement of K^+ and glucose into the cell or calcium administration which antagonizes the cardiotoxic effects of potassium. Ideally, electrolytes are monitored as frequently as dictated by the patient's condition. Staff should be familiar with the electrocardiographic changes of hyperkalemia. The signs are peaked T waves bradycardia, decreased P wave amplitude, prolongation of the PR interval, and widening of the QRS complex.

Management of Anuria/Oliguria

Diuretics are indicated if diuresis has not occurred following volume restoration. Furosemide is a loop diuretic, meaning it acts primarily in the ascending loop of Henle. It is a renal vasodilator and a natriuretic (promotes the excretion of sodium) agent. If used in anuria high does should be utilized. If diuresis does not occur then another diuretic or combination of diuretics is used. If the patient is oliguric, then low doses are used and titrated to effect. Furosemide has the potential for potentiating nephrotoxic effects of certain drugs (aminoglycosides) and therefore should not be used in those cases. The technician should be aware that furosemide could cause excessive diuresis and dehydration, hypovolaemia, hypotension, hyponatraemia, metabolic alkalosis, vomiting and diarrhoea.

Mannitol has osmotic diuretic effects, it increases blood volume, and renal perfusion, it works in the glomerulus of the nephron, therefor it will only be effective if the patient is oliguric rather than anuric. It is usually given as a slow bolus over 20 minutes. Significant diuresis should be seen within 30 min, if not it may be repeated once more. Because it contributes to hyperosmolality and ECF edema, its use is discontinued if it fails to work. If significant diuresis occurs, then mannitol may be given as an intermittent bolus every 4–6 hours over the next 24–48 hours or may be administered as a constant infusion.

In low doses, dopamine has been used to increase the renal blood flow in dogs, but its efficacy is dubious. There is little or no documentation on the efficacy of dopamine in dogs and cats with AKI. Its routine use to increase urine production in oliguric or anuric AKI cannot be justified.

These drugs may be used independently or in conjunction with each other. There are no specific guidelines that dictate which order the drugs are used.

Dialysis

If anuria persists, then peritoneal or hemodialysis is considered. Both options are expensive and time consuming. Peritoneal dialysis uses the principles of diffusion, osmosis and ultrafiltration across the peritoneal membranes. A slightly hypertonic dialysate solution is instilled into the patient's abdomen and allowed to dwell for a short period of time (one hour). The patient's peritoneum acts as a semi-permeable membrane to allow the removal of undesirable solutes via diffusion.

Supportive Care

Uraemic patients are prone to vomiting, therefore, centrally acting antiemetics such as metoclopramide, maropitant (Cerenia), or ondansetron (Zofran) may be indicated. Because patients are also at risk for the development of oesophagitis, and ulcerative gastritis drugs that inhibit gastric acid production may be beneficial, including histamine receptor antagonists, such as famotidine and proton pump inhibitors, such as omeprazole (Losec) and gastrointestinal protectants (Sucralfate). Routine nursing principles apply as far as performing physical therapy in the recumbent patient and keeping the patient warm, clean and dry. IV and urinary catheter care should be performed every 48 and 8 hours respectively. The patient should be observed for potential risk factors such as fluid overload, adverse drug reactions, infection, and catheter related problems.

Physiological monitoring should include blood pressure (patient may be hyper or hypotensive), central venous pressure (patient may be hypo or hypervolemic), ECG (looking for arrhythmias or signs suggestive of hyperkalemia), and "ins and outs" and / or frequent body weight to assess fluid balance.

Nutritional Support

Dietary intake is compromised because of anorexia, vomiting and nausea. These patients may be hypercatabolic, as a result nutritional support will be required. Enteral and parenteral feeding has been utilized. Patients should be fed a diet that is comparable to the chronic renal failure patient. The diet is high energy and moderate in protein. There are a variety of commercial prescription diets that meet these requirements and can be blended for tube feeding.

MANAGING URINARY OBSTRUCTIONS

Introduction

The most commonly seen urinary tract emergencies include urinary tract obstruction, leakage of urine due to trauma and acute renal failure. Any of these conditions can lead to life-threatening renal dysfunction, and the patient is likely to need a period of stabilisation prior to sedation or general anesthesia to allow urinary tract catheterisation, urinary diversion techniques or peritoneal drainage and dialysis. Other emergency or critical care patients without urinary tract disease may also require urinary catheterisation. This may be for accurate measurement of urine output to allow assessment of renal perfusion, or simply to prevent urine scalding in the recumbent patient. Urinary catheters or diversion tubes need to be connected to a ‘closed’ collection system; this reduces the risk of ascending infection in the susceptible debilitated patient.

Stabilisation

Urinary tract obstruction or leakage of urine will usually result in azotaemia, hyperkalaemia, acidosis and often hypovolaemia. Hypovolaemic animals need volume resuscitation with an isotonic crystalloid to correct hypoperfusion. Fluid boluses appropriate to the degree of hypovolaemia present should be administered, and any further requirements determined by response to treatment. Hyperkalaemia can be life-threatening. The rising potassium levels have an effect on the myocardium and cause cardiac arrhythmias; animals are often recumbent and semi conscious. If a bradyarrhythmia is detected, an electrocardiogram (ECG) should be performed (see Chapter 10). Correcting hypoperfusion and establishing urine drainage will reduce potassium levels, but often additional treatment is required. This is especially the case where sedation to relieve an obstruction and place a urinary catheter is required. The cardiotoxic effects of hyperkalaemia greatly increase anaesthetic risks. Calcium gluconate 10% solution administered slowly by intravenous injection (0.5– 1.5 ml/kg) is very useful. While it has no effect on serum potassium levels, it stabilises the threshold potential of the myocardial cells; the effect lasts for 20–30 minutes. For cases where the hyperkalaemia is likely to be ongoing (e.g. in urinary tract trauma or acute renal failure), intravenous neutral insulin and glucose can be administered. The resulting uptake of glucose and potassium into cells reduces serum potassium concentrations.

Urinary tract obstruction

In most cases urinary obstruction occurs at the level of the bladder or urethra, although occasionally a ureter can become blocked. The obstruction is most commonly due to uroliths (see Figure 12.2), or urethral plugs in cats, although neoplasia and granulomatous lesions can also cause a blockage. Cats with feline lower urinary tract disease may present in a similar fashion, whilst there may be no physical obstruction, the pain and muscle spasm associated with the condition may lead to a ‘functional’ obstruction. Owners often report stranguria, dysuria or anuria.

Females are less commonly presented than males, due to having wider, shorter urethras. The clinical condition of the patient depends on the duration of obstruction and whether the obstruction is complete or partial (the animal being able to pass some urine). Although the animal will be showing discomfort, systemic signs may not be apparent in the first 24 hours until azotaemia develops. Careful palpation of the abdomen will usually reveal a large tense bladder; care must be taken not to cause rupture. Following stabilisation, sedation or anaesthesia is usually necessary to enable a urinary catheter to be passed and the obstruction relieved. As with all critical animals, the minimum possible dose of sedative or anaesthetic agent should be used.

Agents are selected to minimise the effect on the cardiovascular system. In male dogs, uroliths most commonly lodge at the narrowing of the urethra just proximal to the os penis. Most uroliths lodged in this position can be 'hydropulsed' (flushed with saline) back into the bladder and removed surgically via a cystotomy later (see Figure 12.3). Once the obstruction has been hydropulsed the catheter can be advance into the bladder to drain it. If the obstruction cannot be dislodged, attempts can be made to pass a very narrow catheter past the obstruction into the urinary bladder as a temporary measure, but usually a pre-scrotal urethrostomy is required to remove the stone surgically. Relief of obstruction in male cats requires urethral catheterisation. Some cases may have an intra-penile obstruction; usually the penis will look cyanotic, and it may be possible to break down the obstruction manually with massage. For catheterisation, a 3 French catheter with open tip is used, and saline flushed through the catheter as it is advanced. In some cases where catheterisation proves difficult, emptying the bladder via cystocentesis can ease catheter placement, as the full bladder is no longer pushing caudally on the urethra. If cystocentesis is necessary, then it should be performed with as narrow a gauge needle as possible to minimise damage to the bladder wall and reduce the risk of urine leakage. If the obstruction cannot be cleared, a tube cystostomy may be necessary (see 'Urinary diversion'). Where a urinary catheter is to be left in place (indwelling), it should be connected to a closed collection system to prevent ascending infection (see Urine collection systems). Where an indwelling catheter is to be placed it is important to minimize discomfort, and trauma from the presence of the catheter. Foley catheters are suitable in the dog and bitch (a stylet is helpful to assist in placing them) as they are soft, and can sit in the bladder neck rather than having a long length of catheter in the bladder. Once the balloon has been filled with saline, the catheter can be pulled caudally to seat the balloon in the bladder neck; this prevents removal of the catheter.

Urine diversion

In some situations it may be impossible to pass a urinary catheter into the bladder: it may be undesirable to have urine enter the urethra, or longer term drainage of the bladder may be required. The urethra may need to be bypassed and a means of urine drainage placed direct into the bladder via the body wall. This technique is known as a tube cystostomy (or pre-pubic catheterisation), this is preferable to repeated cystocentesis while stabilizing a patient pre or post surgery. Tube cystostomy catheters are usually Foley catheters. They can be placed during bladder surgery or via a mini-laparotomy just for this purpose. The catheter is placed through a stab incision in the centre of a purse string suture in the bladder wall, the balloon is then inflated and the purse string tightened (see Figure 12.9). The catheter is exited through a stab incision in the body wall, and the bladder anchored internally with sutures to the body wall (see Figure 12.10). The catheter may then be connected to a closed collection system for continuous drainage of the bladder or the catheter can be capped and intermittent drainage performed. The catheter must remain in place for at least 7 days to ensure strong adhesions between the bladder and the body wall have formed and so prevent leakage of urine into the abdomen after removal. Percutaneous catheter placement systems can also be used (locking loop pigtail catheter, placed via a Seldinger technique), which are placed without laparotomy, through the abdominal wall and into the bladder.

Urine collection systems

Where an indwelling urinary catheter is placed, a closed collection system must be used. Leaving an open urinary catheter to drip urine runs the risk of urine scald to the skin and ascending infection. An indwelling catheter connected to a closed collection system is also preferred to allow accurate measurement of urine output. Closed collection systems can either be commercially available, or an emptied intravenous fluid bag can be used (saline or Hartmann's, not glucose-containing fluids), connected via a sterile giving set (see Figure 12.11). The collection bag is placed below the patient to allow urine to drain by gravity, but avoid placing on the floor to reduce the risk of bacterial contamination. Closed systems should be 'broken' as infrequently as possible. If the bag needs to be emptied, or catheter disconnected, it should be done as aseptically as possible as this is the time of greatest risk for introduction of bacteria into the system. Commercial collection bags have the advantage of having a built-in measurement scale, and can usually be emptied via a tap at the bottom of the bag. This avoids disconnecting and connecting the system, as this is when there is greatest risk of contamination being introduced to the system. Some collecting bags also have an anti-reflux chamber to avoid backward flow from the bag to the bladder.

Monitoring urine output

Urine output is one of the most important indicators of renal function in the critical patient. Normal urine output is 1–2 ml/kg/ hour in the normal animal, but may be reduced in dehydrated patients. Much higher levels of urine output may be seen in patients receiving large volume of intravenous fluids. Urine output should be measured every 2–4 hours. As well as an indicator of renal problems, falls in urine output may be due to pre-renal or post-renal factors. In a hypovolaemic animal, capillary beds in the body may be closed down to conserve circulating volume and ensure perfusion of vital organs. The blood supply to the kidneys is one of the last to be affected, so if urine output is not maintained, greater fluid volumes are required. Post-renal azotaemia and reduction in urine output may be seen in urethral obstruction, or with rupture of the ureter, bladder or urethra leading to a uroabdomen. (But bear in mind animals with ruptured bladders can still appear to urinate normally.)
Urine output can be massive in cases where an obstruction has been relieved; cats frequently have post-obstruction dieresis and urine outputs of 50 ml/kg/hour are possible. Measuring the urine output allows intravenous fluid therapy to be matched to ongoing losses, i.e. matching 'ins' to outs'.

Complications of urinary catheterization

Trauma

Trauma can usually be avoided by using smooth, flexible catheters and good technique. Trauma needs to be avoided as it may predispose the patient to bacterial infection as it will damage normal defence barrier mechanisms. Haematuria caused by trauma will interfere with accurate urinalysis – disease processes may have greatly increased the vascular supply to the bladder and urethra and made it more fragile.

Infection

The urine in the kidneys, ureters and bladder of cats and dogs is usually sterile, whereas the distal urethra, vagina and prepuce normally contain bacteria.
Infection is a risk even with careful catheterisation, as these bacteria may be carried into the bladder. There is an increased risk of iatrogenic infection with repeated catheterisation, and with indwelling catheters. Antibiotics should not be given routinely to patients with indwelling catheters to prevent infection; studies have shown this to lead to the development of resistant infections. It is preferable to wait until the catheter is removed, and then treat any infection on the basis of a culture and sensitivity.

Complications can be avoided by correct technique, and placing the catheter in as aseptic a fashion as possible. Hair should be clipped from around the vulva in dogs and cats, and the prepuce in cats. The area around the vulva or prepuce should be cleaned, and the vulva or prepuce flushed with dilute chlorhexidine or pevidene. If an indwelling catheter is to be left in place, an antibiotic ointment can be placed in the prepuce or vulva to help prevent ascending infection.

Acid-Base Balance

The kidney plays a role in acid-base balance in the body but unlike the lungs which 'blow off' CO_2 and regulated acid-base balance rather quickly, the kidney takes hours to days to elicit an effect. Impaired renal function may mean impaired drug metabolism and excretion making proper drug dosing a challenge. This is because the passage of a drug through cell membranes depends on the pH of the drug's environment. When the pH is altered due to impaired renal function and tubular excretion of hydrogen ion, the drug may behave differently. Prolonged drug effects may be seen in the renal patient.

Anesthesia and the Renal Patient

Patients with urinary obstructions are often debilitated and present with any combination of the following issues; dehydration, hypovolemia, hyperkalemia, azotemia and acid-base abnormalities. The kidney receives 20% of cardiac output and adequate renal perfusion under anesthesia cannot always be predicted. Because of this, blood pressure monitoring is critical in the renal patient. Intravenous fluids are also important because they promote diuresis and minimize hypovolemia and hypotension by increasing stroke volume. These patients can often present with a variety of arrhythmias associated with the hyperkalaemia and …… Drugs used for pre-anaesthetics and induction agents should be carefully selected with the individual patient in mind. Many anesthetic drugs are excreted via the kidney making GFR important. Due to the importance of blood pH and proper drug metabolism, underlying electrolyte and acid-base abnormalities should be corrected prior to premedication and anesthesia. Packed cell volume and total protein, BUN, creatinine, electrolytes (sodium, potassium, chloride, bicarbonate) and blood pressure should be part of the minimum database in addition to a thorough physical exam and detailed medical history. A full chemistry panel and complete blood count plus a blood gas and urine specific gravity are also recommended to evaluate organ function and establish a baseline for serial measurements.