NOT SO CUTE: ACUTE KIDNEY INJURY

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Overview

Acute kidney injury (AKI) is a clinical syndrome, defined as a rapid deterioration in kidney function resulting from injury. Prompt recognition, diagnosis and treatment can help minimize or prevent patients from sustaining permanent renal damage.

Kidney Anatomy

The urinary system, comprised of kidneys, ureters, bladder and urethra, is responsible for waste-product removal in the body. The kidneys are comprised of a cortex, medulla, renal pelvis and nephrons. The cortex is the outermost layer of the kidney and the medulla is the innermost layer of the kidney. The renal pelvis is an expansion of the proximal ureter and is essentially a large collecting channel for urine. The nephron is the structural and functional unit of the kidney. The kidneys are made up of thousands of nephrons that are responsible for carrying out the kidney's basic functions. Each nephron works independently to remove body waste, conserve solutes and produce urine. Each nephron is comprised of even smaller microscopic parts, including Bowman's space, glomerulus, loop of Henle, and proximal and distal convoluted tubules.

Kidney Physiology

The kidneys are vital to life and are responsible for many functions that help maintain overall homeostasis. Their functions include fluid regulation, hormone production, and excretion of metabolic waste products. They maintain the volume and composition of body fluids (water and electrolyte balance), they absorb solutes (proteins, amino acids, glucose), and they remove metabolic waste from the body (urea, uric acid, creatinine). Additionally, the kidneys receive approximately 20-25% of overall cardiac output and help maintain arterial blood pressure. When any of the kidneys' functions become disrupted, there can be systemic consequences.

Urine Production

The nephron is the site at which the filtration process and production of urine occurs. Within Bowman's space lies the glomerulus. The glomerulus is a capillary bed through which all renal blood supply circulates, filtering waste products from the blood. The resultant fluid is known as an ultrafiltrate, which moves throughout the remaining nephron. After water and various solutes are reabsorbed within the nephron, the remaining fluid moves to the renal pelvis as urine.

Urine production is dependent on proper nephron function, appropriate blood supply to the glomerulus, and patency of the urinary tract. In a euvolemic patient, normal urine output in dogs and cats is 1-2ml/kg/hr. If there is a change in any of the factors that affect urine production, the result is abnormal urine output (UOP). The three most common kinds of abnormal urine output are polyuria (UOP > 1-2ml/kg/hr), oliguria (UOP < 1ml/kg/hr), and anuria (lack of urine).

Types of AKI

AKI is characterized by the abrupt (acute) inability of the kidney to regulate fluid balance, electrolyte balance, and acid-base balance, as well as the inability to excrete body waste products.

Azotemia is recognized by abnormally high concentrations of body waste compounds within the blood, primarily blood urea nitrogen (BUN) and creatinine. Azotemia can further be grouped as prerenal, intrinsic renal, and postrenal, and is reflective of the type of AKI a patient has.

Prerenal refers to "before" the kidney, meaning injury is caused by other physiological or hemodynamic factors. This means that the animal is suffering from another disease process in which renal perfusion is compromised, affecting renal blood flow and causing ischemic injury. Examples include hypovolemia, dehydration, cardiac compromise, or vasodilatory diseases.

Intrinsic renal refers to direct damage to the renal parenchyma. Examples include renal ischemia, exposure to toxic agents, or infectious insult.

Postrenal refers to "after" the kidney, meaning there is an obstruction or impediment in the outflow of urine that prevents urine from being eliminated from the body. Examples include urethral obstruction, prostatic disease, urolithiasis, trauma, and neoplasia.

Clinical Signs

Because the clinical signs of AKI can be non-specific, varied, and vague, frequently owners don't recognize a renal illness until the disease process has already been occurring. Often, this means patients present somewhere between the initiation and maintenance phases. Some of the most common clinical signs associated with AKI include lethargy, neurological changes, oral ulceration, gastrointestinal (i.e. anorexia, vomiting, diarrhea), dehydration, varying urine output, and abdominal discomfort.

Diagnosis

Diagnosis is dependent on patient history, physical exam, lab findings, and advanced imaging.

Information that should be asked as part of obtaining a thorough patient history include eating and drinking habits, incidence of GI signs, urination and defecation habits, changes in mentation or behavior, potential exposure to something foreign or toxic, and any recent travel. Physical exam findings that may be indicative of renal compromise include halitosis and/or oral ulceration, dehydration, mental or physical depression, bradycardia, and hypothermia. A hallmark finding on physical exam is small/enlarged, painful kidneys on palpation.

It is important to obtain baseline bloodwork prior to initiating fluid therapy. A PCV/TP will give insight into the patient's volume status. A CBC may show changes in white blood cell counts (indicative of infection or immune compromise) or platelet counts (indicative of potential infectious disease). The biochemical profile will show the severity of azotemia, electrolyte shifts, and changes in glucose levels. A urine sample should be collected, as USG is essential to assess renal concentrating ability. A USG of 1.008-1.012 is consistent with AKI as this range is known as isosthenuria, which reflects renal tubular damage. Urine dipstick may show glucose or protein, which reflects poor renal absorption or indicates renal infection. Urine sediment may reveal many findings; red blood cells (glomerular damage), casts (renal tubular damage), crystals, and bacteria (urinary tract infection).

Abdominal radiographs can give a gross anatomical review off the renal system. The renal silhouette will be able to be visualized as normal, reduced or enlarged. Kidney enlargement is classically seen with AKI. The bladder can also be seen to ensure it's intact. Additionally, radiographs allow review for the presence or absence of stones. Abdominal ultrasound can give more detailed information about the renal system. Kidney size can be evaluated as well as parenchymal changes. Assessment of the renal

pelvis can also give insight, as a dilated renal pelvis can be indicative of a urinary obstruction or pyelonephritis.

Treatment

Once diagnosed, treatment of AKI should be focused on addressing the underlying disease process and providing supportive care to minimize the negative consequences.

Venous access is vital to ensure patent delivery of fluid therapy and medications. When determining what size catheter to place, it is important to keep in mind that AKI patients will likely be hospitalized for many days on supportive IV fluids. In addition to peripheral venous access, it's worth mentioning that placement of a multi-lumen central venous catheter may be beneficial. Central catheters allow for administering multiple fluid types and serial blood sample collection.

Isotonic crystalloids are the primary fluid type for treating all types of AKI, as they have the most similar composition to the patient's extracellular fluid compartment. For prerenal AKI, aggressive fluid therapy should be instigated to reverse the inciting cause of hypovolemia (prerenal is reversible with restoration of adequate perfusion). For intrinsic AKI, fluid therapy should be directed towards addressing the ischemic/toxic/infectious cause. For postrenal AKI, fluid therapy should be supported until kidney parameters return to normal.

When developing the patient's fluid plan, the two calculations to take into consideration are the replacement fluid requirement and the maintenance fluid requirement. Replacement fluid therapy is used to expand intravascular and interstitial spaces in order to maintain hydration. Therefore, replacement fluid therapy should be used in patients who are dehydrated and should be calculated on top of the patient's maintenance fluid requirement over 12-24 hours. The calculation for replacement fluid deficit is: (% dehydration) x body weight in kg x 1000 = fluid deficit (in milliliters). Maintenance fluid therapy takes into account the volume of fluid and amount of electrolytes consumed on a daily basis to keep balance within the fluid compartments. The calculation for maintenance fluid therapy is 40-60ml/kg/day. It is important to note that these formulas are intended to be used as a guide when starting fluid therapy; monitoring perfusion parameters should be used to assess and adjust the patient's fluid needs.

The use of pharmacologic agents to treat specific clinical signs should also be implemented. The use of antiemetics not only prevents vomiting and nausea, but also can alleviate abdominal discomfort from any GI signs. If there's an electrolyte derangement, supplementation may be necessary. If an infection is present, antimicrobials are indicated. If oliguria or anuria persist despite fluid therapy, diuretics may be considered (note there's no evidence in human or veterinary medicine showing diuretics improve outcome). Analgesics should also be considered.

In the case of urethral obstruction, rapid stabilization should be provided to relieve the urinary obstruction. Sterile placement of a urinary catheter should occur as soon as possible. Catheter placement should be verified by a lateral radiograph, secured in place, and connected to a sterile closed collection set to be able to quantify UOP.

Although often not financially or geographically available, dialysis or renal replacement therapy is considered the gold standard of AKI treatment. The use of either of these modalities is the most efficient means of managing uremia, acid-base abnormalities, electrolyte derangements and fluid balance in AKI patients. Dialysis is the process of transferring water and solutes from one compartment

to another by means of diffusion across a semipermeable membrane (SPM). The dialysis options available in veterinary medicine include peritoneal dialysis (PD), intermittent hemodialysis (IHD) and continuous renal replacement therapy (CRRT). In peritoneal dialysis, the peritoneum serves as the semipermeable membrane between the peritoneal cavity and the blood within the peritoneal capillaries. Dialysate (mixture that passes through the membrane) is prescribed to maximize elimination of uremic toxins, prevent depletion of normal blood solutes, replenish depleted solutes and minimize physiologic and metabolic disturbances during and after dialysis sessions. PD is accomplished by instilling the dialysate mixture into the abdomen through a peritoneal catheter. The dialysate is allowed to dwell for a prescribed period of time, then is drained into a waste bag. During the instilling/dwelling/draining period, fluid and solutes (primarily waste products- urea and creatinine) are drawn across the membrane through diffusion (solutes move across a SPM from an area of high to low concentration) and convection (movement of solutes with the flow of fluid). Intermittent hemodialysis is the removal of a patient's blood to be run through an artificial kidney machine (dialyzer). Uremic toxins are removed by diffusion across a SPM within the dialyzer (blood is circulated on one side; dialysate is circulated on the other). The entire blood volume of the patient is treated and then returned. IHD takes 4-6 hours per treatment, with treatments done a set number of times per week. Continuous renal replacement therapy is essentially the same process as IHD except that it is done continuously. PD, IHD and CRRT are all considered temporary therapies to replace the function of the kidneys, giving them time to heal. Of these options, PD is the treatment modality that can be performed in any critical care, 24-hour facility, and should be considered if or when conventional AKI therapy is failing or has failed.

Nursing Care of the Renal Patient

Since the kidneys cannot process fluids normally, patients are more susceptible to complications. Close monitoring by the nursing team is essential for patient care and outcome, as these patients are often hospitalized for several days.

Routine assessment of mentation, heart rate (HR), pulse rate, respiratory rate (RR), temperature, mucous membrane (mm) color, capillary refill time (CRT), and blood pressure are all important. These parameters are related to adequate perfusion, and evaluation of them is necessary to help guide goal-directed fluid therapy. Changes in mentation should be closely monitored to ensure patient progress and stability. Respirations also need to be more closely monitored as changes in RR can be indicative of fluid overload. Blood pressure (BP) should be taken every 4-8 hours, as BP is the product of cardiac output (of which the kidneys receive 20-25%) and systemic vascular resistance (amount of blood flow resistance). BP measurements can be taken indirectly, using oscillometric or Doppler method. To ensure consistency, the same limb and cuff size should be used each time a reading is taken. Electrocardiogram (ECG) should also be used if the patient has an arrhythmia; the severity of the arrhythmia and the rate at which is resolves will determine the frequency of ECG monitoring (as often as continuous or intermittently at 4-12 hour intervals). Lastly, the patient's initial body weight and what scale was used (i.e. hallway scale, treatment scale, baby scale) should be documented every 4-8 hours.

When monitoring fluid balance, careful recording of the amount of fluids in and amount of fluids out must be done. Fluids in include IV crystalloids, IV medications, and oral intake. Fluids out is essentially urine output. The most reliable method of quantifying UOP is via a urinary catheter. It's important to maintain sterile technique when handling the urinary catheter and collection set; this includes wearing gloves, using alcohol wipes if accessing urinary ports, and performing urinary catheter care every 8 hours. When recording a patient's UOP, it's helpful to get in the habit of recording it three ways; total UOP, ml/hr UOP, and ml/kg/hr UOP. Other methods of quantifying UOP can be done by collecting

voided samples or weighing diaper pads (1g = 1ml). All UOP should be documented and reported to the attending DVM.

Signs of fluid overload include serous nasal discharge, increased RR, crackles heard on auscultation, coughing, development of subcutaneous/peripheral edema, chemosis, polyuria, restlessness, or a 10% increase in baseline body weight.

Serial blood work should be performed to evaluate BUN, creatinine, PCV/TP and electrolyte levels. If possible, the same machine should be used to measure BUN and creatinine each time. It will depend on the severity of initial lab values, but blood work is typically performed every 12-24 hours (more frequently if there are severe derangements).

Providing nutrition to renal patients is an important and often neglected consideration. Nutrition should be encouraged in hospital, and if a patient won't eat, enteral nutrition should be considered. The use of nasoesophageal or nasogastric feeding tubes is recommended for short-term nutritional supplementation. Enteral nutrition can be provided as either bolus feedings or a constant rate infusion (CRI) of the patient's energy requirements. The practice of syringe feeding to provide nutrition should be discouraged, as it promotes food aversion, has a risk of aspiration, and diminishes the nurse-patient bond.

Conclusion

Acute kidney injury is the acute decline in renal function that manifests from either prerenal, intrinsic renal or postrenal causes. Regardless of the cause, AKI requires timely supportive care and diligent nursing care to promote positive patient outcome.

References

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