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Fluid and electrolyte management in renal disorders: Best practices in nephrology: Review article

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***Abstract*---Background:** The kidneys play a vital role in maintaining fluid, electrolyte, and acid-base balance. Disruptions in these functions are seen in both acute kidney injury (AKI) and chronic kidney disease (CKD). Effective management of fluid and electrolytes is crucial for patient outcomes in renal disorders. **Aim:** This review article aims to consolidate best practices in fluid and electrolyte management for patients with renal disorders, focusing on acute and chronic conditions. Also, to assist the roles for healthcare providers. **Methods:** A comprehensive review of current literature and clinical guidelines was conducted to outline fluid and electrolyte management strategies. Key areas covered include fluid therapy, hydration assessment, and the use of different fluid types and administration routes. **Results:** The review highlights that individualized fluid therapy is essential for both AKI and CKD patients. Accurate assessment of hydration status is critical, with various methods discussed. Intravenous fluid administration is preferred in hospitalized patients, though alternatives like intraosseous and subcutaneous routes are also noted. Balanced polyionic solutions are recommended for initial resuscitation, while dextrose solutions are used for maintenance. The review also emphasizes the importance of cautious fluid administration to prevent overhydration and its associated risks. **Conclusion:** Optimal management of fluid and electrolytes in renal disorders requires a tailored approach based on the patient's condition. Effective fluid therapy involves not only appropriate choice and administration routes but also continuous monitoring and adjustment to avoid complications such as edema and worsening renal function.

***Keywords*---**renal disorders, fluid management, electrolyte balance, acute kidney injury, chronic kidney disease, hydration assessment, fluid therapy.

Introduction

Due to the kidneys' crucial function in maintaining homeostasis, renal failure can result in significant imbalances in fluid, electrolyte, and acid-base regulation. The primary objective of treatment is to correct these imbalances. Kidney disease is traditionally divided into acute and chronic categories. A decompensation of chronic kidney disease often presents as an acute crisis, and although the

therapeutic approaches are similar, the clinical presentations of acute kidney injury (AKI) and decompensated chronic kidney disease (CKD) can differ markedly. Many individuals with AKI require hospitalization for optimal care, while those with CKD might be managed on an outpatient basis or require hospitalization depending on the severity of their fluid and electrolyte imbalances. Azotemia may occur when renal blood flow is reduced, which can result from factors like hypovolemia, hypotension, decreased cardiac output, or increased renal vascular resistance. This hemodynamic azotemia is often quickly reversible upon correction of the underlying issue [1]. Intrinsic kidney injury arises when damage occurs within the renal parenchyma, which may be reversible or irreversible and can involve the glomeruli, tubules, interstitium, or renal vasculature [1]. Post-renal azotemia occurs when urine flow is obstructed, or urine leaks into surrounding tissues, such as in the case of a ruptured bladder or urethra and can be rapidly corrected by diverting urine through a catheter [1]. Both hemodynamic and post-renal causes of azotemia can, if left untreated, progress to intrinsic renal failure. Despite this, significant kidney disease can exist without azotemia, and fluid therapy is often unnecessary in such cases, especially in well-compensated CKD with mild to moderate azotemia [1].

Fluid Management

Normal fluid losses include both insensible and sensible losses. Insensible losses, such as water loss through respiration, stool, and perspiration, are not consciously noticeable, although sweating is minimal in dogs and cats [1]. Respiratory losses, particularly during excessive panting, can vary in dogs, but on average, 22 mL/kg per day is typical [1]. The primary source of sensible fluid loss in healthy individuals is urine output, with additional losses arising from vomiting, diarrhea, drainage from body cavities, or burns. In healthy animals, these losses are replaced through food and water intake. In ill animals, fluid therapy is necessary to compensate for these losses, particularly when water intake is impaired by conditions like vomiting [1]. In renal disease, urine output is often abnormal, either too high or too low, necessitating individualized fluid therapy to maintain balance [1].

Fluid Therapy for Hospitalized Patients

Various definitions of AKI exist. According to the International Renal Interest Society (IRIS), AKI is graded based on serum creatinine concentration increases, with an elevation of 0.3 mg/dL within 48 hours defining Grade I, with further increases indicating higher stages [2,3]. Early intervention to prevent further renal damage is most effective at the initial stages of injury. Although oliguria or anuria are classical signs of AKI, polyuria can also occur, suggesting a less severe form of injury. Patients with CKD can present with a decompensated uremic crisis, which may represent AKI superimposed on chronic disease [2,3]. Several drugs have been investigated for their potential benefit in treating AKI, though their effectiveness varies depending on the clinical context. The most effective treatment strategy for AKI remains the careful management of fluid balance, which involves regular assessment of the patient's hydration status, a personalized fluid treatment plan, frequent reassessment of electrolyte and fluid

balance, and timely adjustments to the treatment plan in response to the rapidly evolving clinical condition of patients with kidney disease.

Hydration Assessment

A critical aspect of developing an appropriate fluid treatment plan is accurately determining hydration status. Blood volume can be measured using methods such as indicator dilution techniques, radioactive tracers, and bioimpedance spectroscopy. However, these precise measurements are not practical in typical clinical settings [4]. Although objective data may be limited, there are several approaches to estimate hydration. Dehydration results from a deficit in the extravascular fluid compartment (interstitial and intracellular spaces), and severe dehydration can lead to intravascular deficits, which may impair perfusion. Clinical signs of dehydration vary by severity [5]. Overhydration, on the other hand, may present with symptoms such as moist mucous membranes, increased skin elasticity, shivering, vomiting, restlessness, nasal discharge, chemosis, tachypnea, cough, pulmonary edema, pleural effusion, ascites, diarrhea, or subcutaneous edema, particularly around the tibiotarsal joints and intermandibular area [6].

Interpreting physical findings related to hydration can be challenging. For example, patients with uremia often experience xerostomia, which causes dry mucous membranes regardless of their hydration status. Conditions such as hypoalbuminemia or vasculitis may lead to interstitial fluid accumulation despite a deficit in intravascular volume. In addition, factors like emaciation or advanced age can reduce skin elasticity, further complicating assessments [6]. An accurate body weight, recorded prior to illness, is an essential tool for evaluating hydration. Monitoring body weight 3 to 4 times daily using the same scale allows for tracking fluid balance. An ill animal may lose 0.5% to 1.0% of its body weight per day due to anorexia; any greater change is likely due to fluid loss or gain [7,8,9]. Blood pressure trends are also useful, as increased blood pressure may indicate fluid gain, while a decrease may suggest fluid loss. In hypertensive patients—80% of dogs with severe acute uremia and 20%–30% of dogs and cats with CKD—observing trends, rather than relying on absolute values, is more informative for assessing hydration. Similarly, changes in packed cell volume (PCV) and total solids may reflect volume changes when bleeding or transfusion are not involved. Since each of these parameters is influenced by factors beyond hydration status, a comprehensive evaluation is necessary. While central venous pressure (CVP) has historically been used to assess intravascular volume, it no longer reliably predicts fluid responsiveness and is therefore not recommended by current guidelines [10,11].

Fluid Administration Routes

In most hospitalized patients, the intravenous (IV) route is the preferred method for fluid administration. However, in certain cases, such as neonates, young puppies, or kittens, IV catheterization may be difficult. In such instances, intraosseous fluid administration can be a viable alternative. In dehydrated patients, fluids administered intraperitoneally are absorbed effectively, but this method is unreliable for inducing diuresis or for managing oliguric patients.

Subcutaneous fluid administration may result in slow or incomplete absorption, and large volumes cannot be administered this way, making it unsuitable for hospital settings. However, it may still have a role in outpatient care.

Type of Fluid

For initial volume resuscitation and dehydration replacement, a balanced polyionic solution like lactated Ringer's solution (LRS), Plasmalyte-148, or Normosol-R is recommended. In cases of hyperkalemia, 0.9% NaCl, which lacks potassium, is a suitable option. After rehydration, fluids with lower sodium concentrations, such as 0.45% NaCl with 2.5% dextrose or one-half strength LRS with 2.5% dextrose, are more appropriate for maintenance. While dextrose 5% in water (D5W) is generally unsuitable as a standalone fluid, it can be mixed with LRS or 0.9% saline to create reduced sodium solutions for specific needs. In cases of hypoalbuminemia, where protein-losing nephropathy, vasculitis, gastrointestinal losses, or bleeding may occur, the administration of crystalloids can lead to interstitial edema due to a lack of colloid oncotic pressure. While colloid solutions, such as hydroxyethyl starch, have been used in these situations, concerns regarding their potential to increase the risk of acute kidney injury (AKI) remain. The role of colloids in AKI treatment is still unclear. Although colloids are sometimes described as "volume-sparing," research has shown that the total fluid volume administered during hospitalization does not differ significantly between patients receiving colloids and those receiving crystalloids. Human albumin is a potential alternative to synthetic colloids, but it carries a risk of anaphylaxis, and canine albumin is currently unavailable.

Anemia is another concern in both acute and chronic kidney disease, with shortened red cell survival due to the uremic environment, blood sampling losses, suppressed erythropoietin production, and possible gastrointestinal bleeding. Rapid blood loss can result in hypotension and hypovolemia, necessitating the quick administration of crystalloids or synthetic colloids. Red blood cell transfusion may be required in cases of symptomatic anemia, though care must be taken in patients with compromised cardiovascular function to avoid congestive heart failure by administering transfusions more slowly than usual [12,13]. Water administered enterally is sometimes overlooked as a fluid source. While vomiting often prevents uremic patients from consuming water voluntarily, water can be delivered via a feeding tube and should be included in fluid calculations. Ultimately, fluid choice should be guided by ongoing monitoring of the patient's fluid and electrolyte balance, especially regarding sodium concentration. Rapid changes in sodium levels can cause central nervous system (CNS) dysfunction, so sodium correction must be gradual and proportionate to the rate at which the imbalance developed [12,14-18].

Volume and Rate

Traditionally, fluid therapy has focused on correcting dehydration by "pushing" fluids to force diuresis, followed by tapering fluids prior to discharge. If azotemia fails to improve, fluid rates are often increased. However, recent evidence suggests that this approach may actually impair renal function by worsening kidney outcomes. Administering crystalloid fluids quickly leads to their distribution into

the interstitium, resulting in interstitial edema, which can hinder oxygen and metabolite diffusion and further impair kidney function [13,14,19]. A revised approach to fluid therapy involves three phases: (1) acute resuscitation to restore intravascular volume, tissue oxygenation, and organ perfusion, (2) maintaining volume homeostasis without fluid overload, and (3) removing unnecessary fluid volume during recovery. This phased approach may improve outcomes by preventing fluid overload and promoting renal recovery [19,20].

Acute Resuscitation

In cases where patients present with hypovolemic shock, which can manifest as dull mentation, hypotension (systolic blood pressure <80 mm Hg), poor peripheral perfusion, hypothermia, or tachycardia, immediate fluid resuscitation is critical to prevent irreversible organ damage. Standard crystalloid dosages are 60-90 mL/kg for dogs and 45-60 mL/kg for cats, administered in one-fourth doses over 5-15 minutes. If hemodynamic parameters do not improve after the first dose, additional doses should be given until the patient is stabilized. Successful resuscitation helps reduce renal morbidity by achieving specific clinical goals [21]. For dehydrated patients, the fluid deficit is calculated using the formula: body weight (kg) × estimated % dehydration = fluid deficit (L). If a fluid bolus is used for initial resuscitation, its volume should be subtracted from the total deficit. The rate of fluid replacement depends on the clinical situation. In cases of acute dehydration due to AKI, rapid replacement over 0-6 hours is advised to restore renal perfusion and prevent further kidney damage. If diminished urine output is suspected, the deficit should be replaced over 2-4 hours to determine whether oliguria is related to volume depletion or a pathologic condition. In patients with compromised cardiac function, a slower rate (6-12 hours) may prevent congestive heart failure. For chronic dehydration, a slower rehydration over 12-24 hours is generally safer to avoid rapid electrolyte shifts or cardiac issues [22-23].

Maintenance Therapy

Once resuscitation has restored euvolemia, the goal of maintenance therapy is to sustain intravascular volume without fluid overload. Traditional methods advocating increased fluid to "flush out uremic toxins" may lead to complications such as edema and organ dysfunction. Excess fluid can accumulate, leading to intrarenal edema, which compresses renal blood vessels and worsens kidney function. Overhydration has been linked to negative outcomes in both human and veterinary patients [24-25]. A starting point for calculating fluid administration involves maintenance fluid rates, often based on body weight. For patients with AKI, fluid therapy should consider actual urine output and ongoing losses, adjusting for conditions like vomiting or polyuria. Following initial resuscitation, fluid volume is typically calculated by adding maintenance fluid (66 mL/kg per day) plus replacement of dehydration and any ongoing losses. Fluid rates should be adjusted based on the patient's response and urine output [26]. Oliguria is often defined as urine output less than 0.25-0.5 mL/kg per hour, with anuria indicating no urine production. Polyuria occurs when urine output exceeds 2 mL/kg per hour. In patients with reduced urine output (oliguria or anuria), the "ins-and-outs" method can guide fluid administration, ensuring appropriate replacement of insensible loss (fluid lost via respiration and stool), actual urine

volume, and ongoing losses. This method is only applied after rehydration has been achieved. Anuric patients should receive fluid only to replace insensible losses, and all fluid therapy should be withheld in cases of overhydration. If fluid therapy fails to induce diuresis in oliguric or anuric patients, dialysis may be the only effective treatment option [26].

Conversion of Oliguria to Nonoliguric:

Reduced urine output can stem from hemodynamic, intrinsic renal, or post-renal factors. In response to hypovolemia or hypotension, the kidneys may retain fluid, leading to a decrease in urine output. Before classifying oliguria as pathological or physiological, it is crucial to optimize renal perfusion through adequate hydration. Administering a fluid volume equal to 3-5% of body weight is recommended for patients who seem hydrated, as dehydration less than 5% is clinically undetectable. However, in patients with apparent fluid overload, fluid administration is unnecessary. While healthy kidneys can regulate blood flow within perfusion pressures ranging from 80 to 180 mm Hg, impaired kidneys may exhibit more linear perfusion responses. Mean arterial pressure should be maintained above 60 to 80 mm Hg, or systolic pressure between 80 to 100 mm Hg, when measured using Doppler technology. Before attributing the lack of urine production to intrinsic renal damage, potential causes like urinary tract obstruction or leakage into peritoneal, retroperitoneal, or subcutaneous tissues should be ruled out.

There is no evidence that diuretics used to convert oliguria to nonoliguria improve the prognosis of acute kidney injury (AKI). In humans, increased urine output due to diuretics may delay dialysis referrals, sometimes inappropriately. However, in veterinary practice, where dialysis is less accessible, increased urine output from diuretic use may allow the administration of additional medications or nutrition, which can be justified even if renal function is not improved. Mannitol, an osmotic diuretic, expands extracellular volume, which may enhance glomerular filtration rate (GFR) and prevent sodium reabsorption in the kidney by inhibiting renin. Mannitol also increases tubular flow, possibly alleviating obstructions from casts and debris. It further reduces vascular resistance and cellular swelling, increases renal blood flow, and scavenges free radicals. Despite these potential benefits, no randomized studies have demonstrated better clinical outcomes with mannitol use compared to volume expansion alone in humans or healthy cats [5,7,29,30].

Mannitol is administered as a slow IV bolus (0.25 to 1.0 g/kg). If urine output increases, a continuous infusion (1 to 2 mg/kg per minute IV) or intermittent doses (0.25 to 0.5 g/kg every 4 to 6 hours) can follow [5]. Higher dosages (2 to 4 g/kg per day) may cause AKI, and it should not be used in dehydrated patients as it exacerbates intracellular dehydration. Conversely, mannitol is also contraindicated in overhydrated patients as it may worsen pulmonary edema. In cases where mannitol is unavailable, hypertonic dextrose can act as an osmotic diuretic. Administering 22 to 66 mL/kg of a 20% dextrose solution can lead to hyperglycemia and glucosuria [32]. Loop diuretics like furosemide increase urine output without enhancing GFR [29,31,33-35]. While they increase urine flow, they do not improve AKI outcomes, indicating that patients who respond likely

have less severe kidney damage [29,33-36]. Nonetheless, loop diuretics are often used despite a lack of proven efficacy, as they carry a low risk of complications. These diuretics inhibit the Na+/2Cl−/K+ pump in the loop of Henle, reducing sodium transport and oxygen consumption, which could protect against further renal injury [36,37].

Although there are theoretical reasons to use loop diuretics, retrospective studies in humans have shown an increased risk of death or failure of renal recovery in the furosemide treatment group, possibly due to delayed recognition of AKI severity or preferential use in severe cases [28,33]. In veterinary settings, loop diuretics may play a more significant role since dialysis is less accessible. Furosemide can treat overhydration or hyperkalemia, but it should not be given to patients with aminoglycoside-induced AKI [5,7]. Urine output should increase within 20 to 60 minutes after IV administration (2 to 6 mg/kg), but high doses (10 to 50 mg/kg) may cause side effects, such as ototoxicity in humans and adverse reactions in animals [7]. A lack of response to high doses indicates therapy should be discontinued, while positive responses allow for repeat dosing every 6 to 8 hours. Continuous rate infusions offer sustained diuresis at lower cumulative doses [33]. Although dopamine has been shown to convert oliguric patients to nonoliguric, it does not improve GFR or patient outcomes, and its adverse effects have led to it no longer being recommended for oliguric AKI except for blood pressure control [29,35,42,43]. Fenoldopam, a selective dopamine-1 receptor agonist, shows potential benefits by increasing cortical and medullary blood flow without the adverse effects of dopamine, though further clinical trials are needed [29,38]. Calcium channel antagonists have also shown potential in reversing renal vasoconstriction and reducing tubular damage after transplantation, though further research is required [48]. Atrial natriuretic peptide (ANP) has been shown to increase GFR by dilating afferent arterioles and constricting efferent arterioles, but clinical trials have yet to demonstrate consistent benefits [29].

Monitoring Fluid Therapy

Ongoing monitoring of fluid status is essential and must be repeated several times daily. This includes assessing physical examination findings and body weight at least twice daily and adjusting the fluid plan as necessary. Monitoring blood pressure is also vital, as is tracking urine output and other fluid losses, which should be correlated with overall volume status. Urine volume can be determined through various methods, such as the use of an indwelling urinary catheter with a closed collection system, collecting naturally voided urine, utilizing a metabolic cage, or weighing cage bedding or litter pans (where 1 mL of urine equals 1 gram). The most accurate method typically involves an indwelling catheter, though challenges such as urine leakage or accidental disconnection can lead to inaccurately low measurements. The risk of iatrogenic urinary tract infections due to catheterization can be minimized through meticulous catheter and patient hygiene, including cleaning the catheter's external parts with antiseptic several times a day and changing the collection bag and tubing daily [49]. In some patients, complete collection of voided urine may be difficult due to lack of cooperation or urinary incontinence, especially in recumbent or obtunded individuals. In such cases, an accurate scale is necessary for measuring small urine volumes in smaller animals, while weighing bedding or litter pans before

and after use may provide a non-invasive method for assessing urine volume in some patients. Fluid losses due to vomiting and diarrhea are typically estimated, while other sources of fluid loss, such as body cavity drainage or nasogastric tube suctioning, can be measured.

Discontinuing Fluid Therapy

In cases of acute kidney injury (AKI), once diuresis is established, polyuria may become significant. Monitoring urine output to prevent inadequate fluid administration is crucial in this context, as well as in cases of oliguria or anuria. Weaning patients off intravenous (IV) fluids is a critical step in management. Once azotemia has either resolved or plateaued, fluid volume can be reduced by 25% daily. If the urine output decreases accordingly without a return of azotemia, tapering should continue over the next 2 to 3 days. However, if urine output remains unchanged, the kidneys may be unable to regulate fluid balance, and further fluid reduction could lead to dehydration. In such cases, attempts to taper fluids should be postponed for several days, and a slower reduction rate (10%–20% daily) should be attempted. In rare instances, it may take weeks for kidney function to return to normal fluid regulation. For patients who do not enter a polyuric phase, the tapering process allows time for any accumulated excess fluid to be excreted. In chronic kidney disease (CKD) cases, once the hemodynamic aspect of azotemia has resolved, serum creatinine concentrations typically decrease by at least 1 mg/dL per day and should be monitored every 48 hours. When the creatinine levels stabilize, indicating no further decrease despite continued IV fluid therapy, fluids should be gradually tapered in preparation for patient discharge. A gradual reduction in fluid therapy over 2 to 3 days is advised to taper aggressive diuresis.

Outpatient Fluid Therapy

While subcutaneous fluid therapy is commonly used in managing kidney disease, its effectiveness has not been rigorously evaluated. Chronic dehydration or ongoing signs of uremia are valid reasons for administering subcutaneous fluids long-term. The dosage is based on subjective assessment of the patient's hydration status and well-being. In cats, a typical starting dose is between 100 to 150 mL daily or every other day. Cats generally appear to respond more favorably to subcutaneous fluid therapy compared to dogs. Suitable fluid choices include lactated Ringer's solution or 0.9% saline, while dextrose-containing fluids carry an increased risk of abscess formation. Many owners can be trained to administer the fluids at home using a fresh needle for each dose. While an administration tube can be implanted in the subcutaneous space to avoid the need for a needle, this method poses a greater risk of infection at the exit site and can lead to subcutaneous fibrosis, resulting in pain during administration and reduced fluid capacity.

Nutritional Support

Renal failure is a highly catabolic condition, and while it is difficult to directly attribute outcomes to nutritional management, poor nutritional status significantly contributes to increased morbidity and mortality in patients . Early

enteral feeding is beneficial in maintaining gastrointestinal mucosal integrity. While renal diets with restricted phosphorus and limited high-quality protein are typically recommended for chronic kidney disease (CKD), an ideal diet for acute kidney injury (AKI) has not yet been identified. In the absence of specific guidelines, enteral diets designed for critically ill animals or humans are often used. Anorexia is a frequent issue in hospitalized patients with kidney disease. If appetite does not improve within a few days of treatment, placing a feeding tube is strongly recommended to ensure the patient receives the necessary caloric intake and to facilitate the administration of oral medications. If vomiting persists and cannot be controlled, partial or total parenteral nutrition (PPN or TPN) may be required. For patients who are anuric or oliguric, the volume of nutritional support, whether enteral or parenteral, may be limited. Most liquid diets suitable for nasoesophageal or nasogastric tubes have a caloric density of approximately 1 kcal/mL. To meet 100% of basal energy requirements, the volume of intake will typically be about double the insensible fluid requirements. Common formulas for calculating TPN also result in nearly twice the insensible fluid requirements. In oliguric patients, the need for nutritional support may necessitate fluid removal via dialysis.

Nutritional Support for Chronic Kidney Disease (CKD)

Chronic Kidney Disease (CKD) management involves specific dietary considerations that aim to reduce the progression of the disease and manage its symptoms.

1. **Diet Composition**:
    - **Phosphorus Restriction**: High phosphorus levels can exacerbate kidney damage. CKD diets often feature reduced phosphorus content to help control blood phosphorus levels and prevent secondary hyperparathyroidism .
    - **Protein Moderation**: Although protein is essential, excessive protein can increase the kidneys' workload and exacerbate renal damage. CKD diets generally include high-quality, moderate-protein sources to meet the animal’s needs while minimizing uremic toxin production .
    - **Increased Omega-3 Fatty Acids**: These are sometimes included to help reduce inflammation and provide renal protective benefits .
    - **Low Sodium**: Reduced sodium helps manage hypertension and fluid retention, which can be associated with CKD.
2. **Dietary Goals**:
    - **Maintain Adequate Nutrition**: Despite the restrictions, it is crucial to provide sufficient calories and nutrients to prevent malnutrition and maintain overall health.
    - **Preserve Renal Function**: Proper diet helps in reducing the progression of kidney disease by minimizing the accumulation of harmful waste products in the blood.
    - **Manage Clinical Symptoms**: Addressing symptoms such as proteinuria and hypertension through diet can contribute to a better quality of life .

3. **Implementation**:
    - **Commercial Renal Diets**: There are specially formulated commercial diets available that meet these nutritional requirements and are typically recommended for managing CKD .
    - **Homemade Diets**: In some cases, homemade diets may be prescribed, but they must be carefully balanced to meet the specific needs of the patient .
4. **Monitoring and Adjustment**:
    - **Regular Assessments**: Frequent monitoring of kidney function parameters, including blood urea nitrogen (BUN), creatinine, and phosphorus levels, is essential to adjust the diet as needed .
    - **Clinical Response**: Changes in the patient's clinical condition or laboratory values may necessitate dietary adjustments to better manage CKD.

By adhering to these dietary principles, CKD patients can potentially experience slowed disease progression and improved quality of life.

Conclusion

Fluid and electrolyte management in renal disorders is a complex yet critical aspect of nephrology. Effective management hinges on a thorough understanding of the underlying pathophysiology of both acute kidney injury (AKI) and chronic kidney disease (CKD). This review underscores the importance of individualized treatment plans, emphasizing that a one-size-fits-all approach is inadequate given the variability in patient responses and conditions. For acute kidney injury, early and precise fluid resuscitation is paramount. The initial focus is on restoring intravascular volume and ensuring adequate tissue perfusion. This is achieved through the careful administration of balanced polyionic solutions. However, the management of AKI does not end with initial resuscitation; ongoing monitoring and adjustment of fluid and electrolyte balance are crucial to prevent fluid overload and worsening renal function. The phased approach—acute resuscitation, maintenance therapy, and careful removal of excess fluid—has shown promise in improving patient outcomes. In chronic kidney disease, fluid management becomes more nuanced. Regular assessment of hydration status, using methods such as monitoring body weight and blood pressure trends, helps guide fluid therapy. While intravenous administration is preferred for hospitalized patients, alternative routes such as intraosseous or subcutaneous administration may be necessary in specific situations. The choice of fluids and their administration rates must be tailored to each patient's condition, considering their overall fluid balance and the presence of any complications. The review also highlights the challenges and potential pitfalls associated with fluid therapy, including the risks of overhydration and the need for careful monitoring to avoid adverse effects such as edema and worsening kidney function. While diuretics and osmotic agents have roles in managing fluid balance, their efficacy and safety must be weighed against potential risks. In conclusion, the review reinforces that fluid and electrolyte management in renal disorders requires a meticulous and individualized approach. Continuous evaluation and adjustment are essential to achieving optimal patient outcomes, minimizing complications, and promoting renal recovery.

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إدارة السوائل والإلكتروليتات في اضطرابات الكلى: أفضل الممارسات في علم الطب- مقالة مراجعة

الملخص:

الخلفية :تلعب الكلى دوراً حيوياً في الحفاظ على توازن السوائل والإلكتروليتات وحمض-قاعدة. يُلاحظ وجود اضطرابات في هذه الوظائف في كل من إصابة الكلى الحادة (AKI) وأمراض الكلى المزمنة .(CKD) تعتبر الإدارة الفعالة للسوائل والإلكتروليتات أمراً حاسماً لنتائج المرضى في اضطرابات الكلى.

الهدف :تهدف هذه المقالة المراجعة إلى توحيد أفضل الممارسات في إدارة السوائل والإلكتروليتات للمرضى الذين يعانون من اضطرابات الكلى، مع التركيز على الحالات الحادة والمزمنة. مع التركيز على افضل الممارسات للعاملين بالقطاع الطبي.

الطرق :تم إجراء مراجعة شاملة للأدبيات الحالية والإرشادات السريرية لتوضيح استراتيجيات إدارة السوائل والإلكتروليتات. تشمل المجالات الرئيسية التي تمت تغطيتها العلاج بالسوائل، تقييم الترطيب، واستخدام أنواع السوائل المختلفة وطرق الإعطاء.

النتائج :تسلط المراجعة الضوء على أن العلاج بالسوائل الفردي ضروري لكل من مرضى AKI و .CKDيعتبر التقييم الدقيق لحالة الترطيب أمراً حاسماً، مع مناقشة الأساليب المختلفة. يُفضل استخدام السوائل الوريدية في المرضى المنومين، رغم أنه تم الإشارة أيضاً إلى البدائل مثل الطرق داخل العظمية وتحت الجلدية. يُوصى باستخدام الحلول المتوازنة متعددة الأيونات للإنعاش الأولي، بينما تُستخدم حلول الدكستروز للصيانة. كما تُشدد المراجعة على أهمية الحذر في إدارة السوائل لتجنب فرط الترطيب والمخاطر المرتبطة به.

الاستنتاج :تتطلب الإدارة المثلى للسوائل والإلكتروليتات في اضطرابات الكلى نهجاً مخصصاً بناءً على حالة المريض. يشمل العلاج الفعال بالسوائل ليس فقط اختيار الطرق المناسبة للإعطاء، ولكن أيضاً المراقبة المستمرة والتعديل لتجنب المضاعفات مثل الوذمة وتفاقم وظيفة الكلى.

الكلمات المفتاحية :اضطرابات الكلى، إدارة السوائل، توازن الإلكتروليتات، إصابة الكلى الحادة، أمراض الكلى المزمنة، تقييم الترطيب، العلاج بالسوائل