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LESS IS MORE: FLUID THERAPY IN RENAL DISEASE

Fluid therapy is the most important therapy to treat and support a patient suffering from acute kidney injury (AKI) or from acute on chronic renal failure (CRF).

However, how much fluid is appropriate for the patient with AKI or CRF is still a very controversial topic. Fluids are often administered to patients with AKI or CRF to restore circulating volume in case of hypovolaemia and to restore and maintain hydration and ongoing fluid losses (i.e vomiting, diarrhoea). About 30–75% of AKI arises from renal perfusion problems, including hemodynamic (pre-renal) disorders and renal ischemia. Fluids in these patients are also used to maintain electrolyte and acid-base status, provide nutrition and medications.

Traditionally, once perfusion abnormalities have been corrected, intravenous fluids at higher rate that necessary to correct existing dehydration or fluid losses have been suggested (forced diuresis) , with the goal of maximizing glomerular filtration rate (GFR), renal blood flow (RBF) and increase urine production. Here resides the first misconception of fluid therapy in AKI or CRF: patients with non-oliguric renal failure have very often a fixed urinary output and are unable to respond to fluid challenge or forced diuresis with increasing their urinary production.

Another common belief is that forced diuresis will help the kidneys to get rid of (to "flush") nephrotoxins and other waste products by enhancing their renal excretions. However, there is no literature data both in human medicine and in veterinary medicine to support this statement. On the contrary, numerous human study have concluded that liberal fluid therapy, or forced diuresis, in patients with AKI or CRF is associated with fluid overload and that fluid overload has severe negative consequences associated to increased morbidity and mortality. Patients with over 10% volume overload are 2–3 times more likely to die than those with less overhydration .

Clinically, fluid overload will manifest as an expansion of the interstitial space and increased venous pressure. The kidney is particularly affected and fluid overload will manifest as congestion and increased venous pressure, which causes increased renal subcapsular pressure and secondary decrease in RBF and GFR . Both renal venous congestion and renal interstitial oedema are considered important factors that initiate and maintain AKI.

The question that remains is how can we better titrate fluid therapy in our patients with AKI or CRF. It is clear that our effort should be focused on maintaining fluid balance homeostasis and/or preventing fluid overload. One of the common reason for fluid overload is the failure to adjust the fluid administration rate based on the actual urinary production of the patient and to titrate it in the face of decreased urine production. Careful calculation ad monitoring of the "ins and outs" (fluid balance) can be used in these patients to provide a guide for the appropriate quantities of fluids to administer. It is important to assess the patient need for fluid administration and consider in the fluid balance all fluids administered as part of maintenance and eventual correction fluid therapy, fluid used as nutrition, to dilute medications. The water and food that the patient is taken should also be considered in the patient's fluid balance. All these fluids make the patient's INS. It is fundamental that we are able to assess the patient's urinary production and for this reason an indwelling urinary catheter should be placed. To decrease the risk of an iatrogenic urinary tract infection , the urinary catheter should be managed with careful attention and max hygiene. If a urinary catheter cannot be positioned , especially in cats, the urinary production and the fluid balance should be evaluated by weighing the litter tray before and after urination and /or by weighing in small dogs the cage bedding or litter pans before and after use (with the assumption that1 ml of urine = 1 g).

If the patient is judged to be oliguric, other factors influencing urinary production have already been assessed and the patient is hydrated and well perfused, then urinary production should be stimulated with the use of diuretics, although it is important to remember that diuretics have never been shown to prevent AKI or improve outcomes. Another important factor to consider when we talk about fluid therapy in AKI or CRF is the type of fluid we use. Different fluid therapy solutions are used in human and veterinary medicine to treat shock and dehydration. These include colloids such as synthetic hydroxyethyl starches (HES) and isotonic crystalloids, including saline solution

NaCl 0.9 %, or buffered isotonic crystalloid solution like lactate-based solutions (i.e. Hartmann's solution, ringer lactate) or acetate based solution (i.e. ringer acetate) Colloid solution have been, especially in human medicine associated with the development of AKI . The proposed mechanism is the development of osmotic nephrosis, where tubular uptake of the colloid molecules by pinocytosis with subsequent storage in vacuoles is the proposed mechanism. The presence of these vacuoles will cause alteration of the cells and of their homeostasis and cell apoptosis and death. Multiple studies have also shown that the administration of 0.9%Nacl saline solution is associated with the development of metabolic acidosis and that its high chloride content may affect kidney function. In particular, elevated chloride anion could lead to a reduction in glomerular filtration rate via a tubuloglomerular feedback mechanism and afferent arteriolar vasoconstriction thereby lowering the RBF and GFR. For these reasons fluid therapy for restoration of volume and hydration in AKI or CRF, should be performed using a buffered crystalloid isotonic solution.

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