Among the non-infectious diseases acquired during hospitalization, acute kidney injury involves an increased risk of mortality for many hospitalized patients.

Acute kidney injury that develops during hospitalization (HAAKI – hospitalization acquired acute kidney injury) is defined as an abrupt decline in kidney function in patients receiving treatment. The International Society of Renal Interest, IRIS, established guidelines that classify acute kidney disease based on the serum creatinine value and urine output. It is now known that small changes in serum creatinine concentration (0.3 mg/dl increase from baseline, even within the non-azotemic range) may involve important clinical consequences.

In human medicine, the most frequent causes are sepsis, critical illness, major surgery, administration of radiological contrasts and aminoglycosides during hospitalization.

In canine patients, the most common cause of HAAKI was exposure to nephrotoxic substances (Behrend et al., 1996). In cats subjected to mechanical ventilation, azotaemia and oliguria were identified causes of mortality (Lee et al., 2005) and also developed in 30% of dogs that underwent mechanical ventilation. The maintenance of PEEP may lead to hemodynamic effects by altering the renal perfusion and the neuroendocrine response.

Drug-induced kidney disease (DIKD) is one of the causes of AKI. Given the complexity of epidemiology in human medicine, it has been proposed to address the DIKD taking into account factors such as risk, recognition of the problem, response to treatment, the need for renal replacement therapy and recovery of renal function.

The risk associated with the administration of a substance must be assessed in relation to specific markers of renal function, dose and interval of administration and interaction with other drugs.

Drug interactions in hospitalized patients are another cause of HAAKI. For example, the administration of antibiotics that inhibit cytochrome P450 together with antihypertensive drugs that are eliminated through this metabolic route can lead to hypotension due to increased plasma concentrations of the drug and as a consequence of triggering acute kidney injury (Gandhi et al., 2013).

Human patients on drugs that block the renin angiotensin axis (ACEi or ARAII) during hospitalization who in addition suffer from chronic kidney disease, develop hypotension or need surgery, have a higher risk of AKI than those who are not treated with these drugs (Alabdan et al., 2017). In addition, canine patients who take ACEi on the same day of surgery may have significant periods of intraoperative hypotension (Coleman et al., 2016).

During hospitalization, the highest risk associated with fluid overload is suffered by patients with low oncotic pressure, compensated and renal occult heart disease, since they have less capacity to compensate for excess fluid. In addition, septic animals may suffer from myocardial depression as well as other cardiovascular alterations that make it difficult for them to tolerate the same volume of fluids as a healthy patient.
The choice of an inappropriate fluid (e.g. hypotonic) can lead to hyponatremia in patients with excessive activation of ADH (antidiuretic hormone) and water retention, or can lead to sodium retention (e.g. isotonic fluid) in patients who, under maintenance conditions, do not need the excessive amount of sodium provided by this type of fluid.

There is a weak but significant relationship between the percentage of fluid retained, the severity of the disease and the risk of death.

Some findings in the physical examination that should alert us about the development of a volume overload are: oedema (hock, intermandibular, distal area of the extremities), nasal discharge, increase in respiratory rate at rest, increase in respiratory sounds, etc.

The early detection of fluid overload can be helped by the daily weighing of patients, calculation of the income and loss of fluids, as well as chest radiographs and echocardiograms (determination of the LA/Ao ratio).

The treatment consists of the cessation or at least limitation of fluid therapy, diuretic administration or dialysis.

Some data in dogs suggest that the administration of colloids may increase the risk of AKI and death. However, the available studies are conflicting and also do not coincide in the criteria to establish the existence of acute kidney injury. In cats, there is no evidence that these fluids may cause AKI. Some precautions to take into account when deciding to use a colloid are the following: to limit its use to acute resuscitation for a maximum of 24 hours, to use algorithms of response to fluids and predefined hemodynamic endpoints, to comply with the maximum dose, to use objective markers of hypovolemia (e.g. lactate) and to avoid them in pre-existing or acute renal failure.

In the study by Goic et al., 2016, an incidence of contrast-induced nephropathy in veterinary medicine of 7.6% was observed (increase of 0.5 mg/dL of creatinine with regard to baseline), and 2.2% of patients showed clinical signs associated to kidney injury. It is not entirely clear why contrast media cause kidney injury but it seems that it is due to a combination of direct toxicity on tubular cells, ischemia, renal arterial vasoconstriction, oxidative damage, etc. This toxicity is also aggravated in case of dehydration or coexisting renal disease. Therefore, prior rehydration plays an important role in reducing toxicity. The characteristics of the contrast medium (osmolality, viscosity and chemical structure) have also been associated with the level of toxicity.

In a recent study it has been observed that human patients who present both hypocalcaemia and hypercalcaemia at the time of admission have a higher risk of suffering acute kidney injury during hospitalization (Thongprayoon et al., 2017).

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