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SPECIAL ARTICLE

Prevention of acute kidney injury in Intensive Care Units

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Abstract Acute kidney injury (AKI) is a growing concern in Intensive Care Units. The advanced age of our patients, with the increase in associated morbidity and the complexity of the treatments provided favor the development of AKI. Since no effective treatment for AKI is available, all efforts are aimed at prevention and early detection of the disorder in order to establish secondary preventive measures to impede AKI progression. In critical patients, the most frequent causes are sepsis and situations that result in renal hypoperfusion; preventive measures are therefore directed at securing hydration and correct hemodynamics through fluid perfusion and the use of inotropic or vasoactive drugs, according to the underlying disease condition. Apart from these circumstances, a number of situations could lead to AKI, related to the administration of nephrotoxic drugs, intra-tubular deposits, the administration of iodinated contrast media, liver failure and major surgery (mainly heart surgery). In these cases, in addition to hydration, there are other specific preventive measures adapted to each condition.

PALABRAS CLAVE
Lesión renal aguda;
Prevención primaria;
Prevención secundaria

Prevención de la lesión renal aguda en las unidades de cuidados intensivos

Resumen La lesión renal aguda (LRA) constituye un problema de importancia creciente en las unidades de cuidados intensivos. La mayor edad de nuestros pacientes, con el aumento de la morbilidad asociada, y la complejidad de los tratamientos realizados favorecen su desarrollo.

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Puesto que la LRA carece de tratamiento eficaz, todos los esfuerzos se dirigen a la prevención y a su detección precoz con el fin de establecer medidas de prevención secundaria que impidan su progresión. En el paciente crítico, las causas más frecuentemente implicadas son la sepsis y las situaciones que provocan hipoperfusión renal, por lo que las medidas preventivas irán encaminadas a mantener un estado de hidratación y hemodinámico correcto mediante perfusión de fluidos y el uso de fármacos inotrópicos o vasoactivos en función de la enfermedad subyacente. Además de estas circunstancias, existen distintas situaciones que pueden favorecer la LRA, relacionadas con la administración de fármacos nefrotóxicos, los depósitos intratubulares, la administración de contrastes iodados, el fallo hepático y la cirugía mayor, fundamentalmente cirugía cardiaca. En estos casos, además de la hidratación, se dispone de otros aspectos preventivos específicos de cada entidad.

Introduction

Acute kidney injury (AKI) is a common problem in Intensive Care Units (ICU) with a high level of associated mortality. Several studies had already demonstrated that even small increases in serum creatinine were associated with a poorer prognosis,[1] considering acute kidney disease as an independent risk factor of mortality. Therefore, the early identification of patients at risk, the application of preventive strategies and carrying out early diagnosis and treatment are fundamental for reducing its incidence. In this chapter we will revise the situations most commonly related to AKI in critical patients, discussing the most appropriate prevention measures. If, in spite of this, AKI becomes unavoidable, efforts should be made to reduce its duration and to achieve the most complete recovery possible of kidney function (secondary prevention).

Primary prevention

Patients at risk

In patients with intrahospital AKI, the cause is usually multifactorial, with kidney hypoperfusion being the most common (fundamentally related to hypovolemia, heart failure and arterial hypotension), followed by the administration of nephrotoxic drugs and thirdly, contrast associated acute kidney injury (CA-AKI).[2] Critical patients, given their differential characteristics, more often experience AKI associated with sepsis and hypovolemia, with nephrotoxic drugs occupying third position.[3] Numerous risk factors have been identified in the literature connected with the development of AKI (Table 1), having an age of over 75 years, Diabetes Mellitus (DM) and the presence of chronic kidney disease (CKD) being the most notable. The most significant clinical factors include sepsis, hypovolemia, arterial hypotension, congestive heart failure, time of clamping in patients who undergo heart surgery, complex and prolonged surgery, a high severity index score at hospital admission and recent administration of nephrotoxic medication.[4] With all of this, different scales have been proposed to stratify patients at risk, although currently these types of tools have yet to be developed.

Table 1 Risk factors for the development of AKI.

Risk factor	Odds ratio (95% CI)
Disease severity	9.08 (4.57–13.60)
Age	4.95 (3.79–6.12)
Use of vasopressors	4.52 (2.03–10.05)
Sepsis/SIRS	4.15 (2.36–7.32)
Hypotension/Shock	3.33 (1.70–6.52)
High risk/urgent surgery	2.34 (1.23–4.49)
Heart failure	2.05 (1.77–2.38)
Diabetes	1.58 (1.36–1.84)
Use of nephrotoxic medication	1.53 (1.09–2.14)
Hypertension	1.43 (1.08–1.89)
Baseline creatinine	0.14 (0.01–0.27)

Modified from Ref. 4.

AKI prevention measures in critical patients

Management of acute illness

With regard to the actual pathologies of the critical patient, sepsis stands out as the main cause of acute kidney dysfunction, with an incidence of 15–20%.[3] Regarding the non-septic AKI, it has a higher mortality rate, a longer stay in the ICU, but a better rate of renal recovery, with similar continuous renal replacement therapy (CRRT) needs.[5] In this clinical context, hypovolemia and hypotension tend to trigger AKI, and although cases have been reported with normal hemodynamic parameters,[6] early resuscitation with fluids[7] and vasoactive drugs are the basis for its prevention and treatment, taking into account that water overload can lead to tissue edema, intraabdominal hypertension, multiorgan dysfunction and greater mortality.[8]

Acute heart failure (HF) is also associated with a higher risk of AKI and a worse prognosis. Type 1 Cardio-renal syndrome (CRS) appears in 27–40% of all acute descompensated heart failure, having a complex physiopathology.[9]

Traditionally, secondary renal hypoperfusion has been attributed to a low cardiac output, however, the results

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