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Levosimendan and Acute Kidney Injury

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Mini Review

Severe sepsis is one of the most common causes of death in intensive care units, while in the presence of septic shock, the mortality rate reaches approximately 70% despite the progress made in the care of critical patients [1]. The widespread inflammatory and procoagulant response caused by sepsis leads to diffuse endothelial dysfunction, endovascular damage, and eventually multiple organ failure. Sepsis, ischemia reperfusion damage, toxic nephropathy, hypovolemia and urinary system obstruction can cause acute renal failure (ARF). However, it was experimentally observed that medullary and cortical blood flow in septic ARF continued or even increased, and this was described as a completely different physiological event from ABY which was not due to sepsis [2].

Major systemic and local mediators, neutrophil-endothelial interactions, microvascular thromboses, renal hypoperfusion, and reperfusion damage have been blamed for the pathogenesis of acute renal failure. Norepinephrine, angiotensin II and vasopressin are important systemic mediators in sepsis. Local mediators, especially tumor necrosis factor (TNF) or interleukin 1 (IL-1), adhesion molecules, oxygen free radicals, catalyzes the if A2(TXA2), prostaglandin E2(PGE2), leukotrienes, platelet-induced growth factor, endothelin, nitric oxide(no) and adenosine include [3]. Nearly half of patients with acute kidney damage (AKD) have sepsis, while in intensive care units, AKD is accompanied by more septic shock. Mortality is higher in patients with sepsis-induced AKD. Adequate fluid replacement, early renal replacement therapy are useful for patients, but there is no method to treat septic AKD [4].

If hypotension cannot be corrected despite fluid resuscitation in sepsis treatment, vasopressor therapy is recommended [5]. Dopamine and norepinephrine were the first vasopressors to be selected in the treatment of Sepsis and septic shock [6]. Levosimendan is a new inotropic and vasodilator agent that has been proven to be beneficial, especially in patients with acute heart failure and acute coronary syndrome. [7]. It opens ATP - sensitive potassium channels in vascular smooth novellas cells, causing arteriolar-venous dilation. This mechanism of action is responsible for coronary, pulmonary, renal and systemic vasodilation [8,9]. In addition to blood urea nitrogen and creatinine, new and specific methods such as cystatin c, neutrophil gelatinase associated lipocalin (NGAL)], IL-8, kidney damage molecule [Kidney Injury Molecule(KIM)]-1 have been introduced in recent years to show acute kidney damage [10].

Neutrophil gelatinase associated lipocalin has been reported as the earliest and most reliable laboratory parameter showing renal ischemia or nephrotoxicity in humans, especially in kidney, lung, stomach and colon cells [11]. NGAL levels can be detected in both urine and plasma within 2-6 hours after AKD [12]. Law et al. [13] they showed that levosimendane reduces tubular necrosis and atrophy in experimental renal ischemia reperfusion damage. However, the study examining the effect of levosimendan on acute kidney damage in polymicrobial sepsis model induced by cecal ligation perforation method was not reached.

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