

Chapter 17

Acute Kidney Injury following Cardiac Surgery

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ABSTRACT

The primary goal of this chapter is to provide the reader with an overview of basic renal physiology and function and to review the identification, pathogenesis, and treatment of acute kidney injury following cardiac surgery. Particular focus will be directed toward the diagnostic criteria for acute kidney injury, short- and long-term impacts on patient outcomes, role of novel biomarkers, mechanisms of acute renal injury, general management principles, preventative strategies, and the influence of anesthetic and surgical techniques on its development. The content of this chapter will serve to underscore a particularly harmful but likely underappreciated problem affecting patients in the cardiothoracic critical care setting.

INTRODUCTION

Fundamental critical care principles include early recognition, stabilization, and prevention of secondary injury. Especially given the aging cardiac surgical population and the heavy burden of coexisting disease this cohort carries, following these tenants is essential to avoid poor outcomes. Consequently this chapter will primarily focus on the early diagnosis, anticipation, and management of acute kidney injury following cardiac surgery. The specific aim is to highlight established prevention strategies and draw attention to emerging diagnostic tools and therapeutic agents.

BACKGROUND

The development of acute kidney injury following cardiac surgery is common and contributes to considerable patient morbidity, mortality, and resource utilization. Given the increasing number of cardiac

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surgical procedures performed and the substantial burden of comorbid disease in this patient population, the search for early markers of injury, specific prevention practices, new pharmacologic therapies, and timely management methods is essential. In line with ongoing research efforts, the future of disease management lies in the avoidance of medications, physiologic states, and iatrogenic procedures that promote renal injury in the perioperative period. Furthermore, the investigation into innovative therapies to prevent injury at the cellular and molecular levels represents the next phase of treatment.

RENAL ANATOMY AND PHYSIOLOGY

The Nephron

The kidney has a number of important physiologic functions including balancing electrolytes to maintain cardiovascular stability and prevention of dysrhythmias; preserving volume status through the regulation of salt and water; maintaining pH by regulating acid-base status for the appropriate functioning of medications, enzymes, and cells; eliminating drug metabolites and toxins; and production of renin, erythropoietin, and calcitriol.

The nephron is the functional unit of the kidney, and each kidney contains approximately 1 million nephrons. The proximal end of the nephron consists of Bowman's capsule surrounding a glomerulus, a network of capillaries supplied by an afferent arteriole and drained by an efferent arteriole. Collectively, this unit is known as the renal corpuscle. The basement membranes of the glomerular endothelial cells form a filter that prevents charged molecules, large proteins, and cells from entering the nephron while allowing ions, amino acids, and water to freely pass. Fluid is initially absorbed from the glomerulus into Bowman's capsule and then subsequently flows through the proximal tubule, loop of Henle, distal tubule, and finally to the collecting duct (see Figure 1). In the proximal tubule, as sodium and water are reabsorbed, organic anions and cations are secreted. Other molecules and ions transported here include glucose, proteins, amino acids, bicarbonate, chloride, potassium, magnesium, calcium, and lactate. Next fluid travels to the loop of Henle where it becomes concentrated, creating a gradient that allows for additional fluid reabsorption via osmosis further down the nephron. Also in the loop of Henle, sodium, chloride, potassium, calcium, and magnesium are reabsorbed. The initial segment of the distal tubule, called the macula densa, functions primarily to monitor the chloride concentration of the passing fluid. Based on this concentration, the macula densa alters afferent arteriolar tone and renin release to regulate changes in the glomerular filtration rate (GFR). The distal tubule also reabsorbs and secretes electrolytes and regulates acid-base status by adjusting the fluid's hydrogen ion and bicarbonate concentration. Finally as the fluid enters the collecting duct, the concentration gradient created by the loop of Henle allows for further water reabsorption and the formation of hypertonic urine (Loutzenhiser, Griffin, Williamson, & Bidani, 2006).

Renal Blood Flow

The combined blood flow through the kidneys consists of approximately 25% of the total cardiac output.

Each kidney typically receives its blood supply from a single renal artery, a branch off the aorta and is drained by a single renal vein which empties into the inferior vena cava. The difference in pressure between the renal artery and renal vein creates a transrenal pressure gradient, which drives the perfusion

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