

MANAGEMENT OF SECONDARY ACUTE RENAL FAILURE IN CRITICAL ILL PATIENT

Essay

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بِسْمِ اللَّهِ الرَّحْمَنِ الرَّحِيمِ

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List of Abbreviations

ACEIsAngiotensin-Converting Enzyme Inhibitors
ADHAnti Diuretic Hormone
AKIAcute Kidney Injury
ARFAcute Renal Failure
ASAAcetylsalicylic Acid
ATNAcute Tubular Necrosis
ATPAdenosine Triphosphate
AVPArginine Vasopressin
BUNBlood Urea Nitrogen
CAHContinuous Arteriovenous Hemofiltration
CAVHDContinuous Arteriovenous Hemodialysis
CAVHDFContinuous Arteriovenous Hemodiafiltration
CHFCongestive Heart Failure
COX IICyclooxygenase II
CRRTContinuous Renal Replacement Therapy
ECFExtracellular Fluid
EDDExtended Daily Dialysis
GFRGlomerular Filtration Rate
HRsHepatorenal Syndrome
HuEpoRecombinant Human Erythropoietin
IHDIntermittent Hemodialysis
IL-18Interleukin-18
IUPIntravenous Pyelogram
JGAJuxtaglomerular Apparatus

List of Abbreviations (Cont.)

KIM-1	Kidney Injury Molecule-1
MRI	Magnetic Resonance Imaging
NGAL	Neutrophil Gelatinase – Associated Lipocalin
NSAIDs	Nonsteroidal Anti-Inflammatory Drugs
PAHA	Para-Amino Hippuric Acid
PD	Peritoneal Dialysis
PEEP	Positive End Expiratory Pressure
RFF	Renal Blood Flow
RRT	Renal Replacement Therapy
SCUF	Slow Continuous Ultra Filtration
SLEP	Slow Continuous Low Efficiency Dialysis

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INTRODUCTION

Acute renal failure (ARF) is rarely an isolated process but is often a complication of underlying conditions such as sepsis, trauma, and multiple-organ failure in critically ill patients. The kidneys remove waste products and help balance water and salt and other minerals (electrolytes) in blood. When the kidneys stop working, waste products, fluids, and electrolytes build up in the body. This can cause problems that can be deadly (*Hoste EA and Kellum JA, 2004*).

Secondary acute renal failure can be due to many different causes. Generally these causes can be divided into two categories. Pre-renal means the cause is before the kidney or glomerulus, this is caused by a decrease in the amount of blood that gets to the kidney, for example heart failure, liver failure and shock. Another class of acute renal failure is post-renal. In this type, there is an obstruction to the flow of urine from the kidney. The most common example is prostate problems in men, urinary tract cancers, which directly obstruct the urine flow, or cancers in the abdomen or pelvis that push on the ureters that carry the urine from the kidney to the bladder (*Bolanos L, et al., 2002*).

Early recognition is critical. Because renal failure is often asymptomatic, it must be detected by carefully

tracking the serum creatinine level. Severe acute renal failure is defined by the following criteria: a) a serum creatinine concentration of ≥ 3.5 mg/dL and/or a blood urea nitrogen concentration of ≥ 100 mg/dL b) an increase in blood urea nitrogen or serum creatinine concentration, such that the concentration is 100% above the baseline value in patients with previous chronic renal insufficiency (serum creatinine concentration of > 1.8 mg/dL, excluding those patients with a basal serum creatinine concentration of > 3.4 mg/dl (*Marenzi G, et al, 2006*).

Acute renal failure is a frequent complication in critically ill patients (up to 20% of critically ill patients) and carries a mortality of 50 to 70%. Depending on the severity and duration of the renal dysfunction, accumulation of metabolic substances is accompanied by metabolic derangements, such as metabolic acidosis, hyperkalemia, disturbances of body fluid balance; and effects on many other organ systems (*Palevsky Paul M, 2004*).

The goals of a preventive strategy of acute renal failure are to preserve renal function, to prevent death, complications (volume overload, acid-base disturbances, and electrolyte abnormalities) and the need for chronic dialysis, with minimum adverse effect (*Fieghen H, et al., 2009*).

Treatment generally is directed at support of blood pressure and flow of the blood to the kidneys. As well, any offending agents should be discontinued and any nephrotoxic agents should be avoided. Some cases will be severe enough to require dialysis to remove toxins from the body until the kidneys can recover. Sometimes, the damage is severe enough that it is irreversible and the patient will require long-term dialysis or renal transplant (*Kozek-Langenecker SA, et al., 2002*).

The outcome of critically ill patients in whom acute renal failure (ARF) develops is still poor. Continuous renal replacement therapy (CRRT) is now increasingly accepted as the preferred treatment modality in the management of ARF in these patients (*Bolanos L, et al., 2002*).

AIM OF THE WORK

The aim of this study is to discuss new update of management of secondary acute renal failure in critically ill patient.

CHAPTER (I): RENAL PHYSIOLOGY

Most people are familiar with one important function of the kidneys - to rid the body of waste materials that are either ingested or produced by metabolism. A second function that is especially critical is to control the volume and composition of the body fluids. For water and virtually all electrolytes in the body, the balance between intake (due to ingestion or metabolic production) and output (due to excretion or metabolic consumption) is maintained in large part by the kidneys. This regulatory function of the kidneys maintains the stable environment of the cells necessary for them to perform their various activities. The kidneys perform their most important functions by filtering the plasma and removing substances from the filtrate at variable rates, depending on the needs of the body. Ultimately, the kidneys clear unwanted substances from the filtrate (and therefore from the blood) by excreting them in the urine while returning substances that are needed back to the blood (*Guyton & Hall, 2006*).

Physiological functions of the kidney:

1. Kidneys play the central role in regulating the water concentration, inorganic-ion composition, and volume of the internal environment. They do so by excreting just enough water and inorganic ions to keep the amounts of these substances in the body relatively constant.

2. Excrete metabolic waste products into the urine as fast as they are produced. This keeps waste products, which can be toxic, from accumulating in the body. These metabolic wastes include urea from the catabolism of protein, uric acid from nucleic acids, creatinine from muscle creatine, the end products of hemoglobin breakdown (which give urine much of its color), and many others.
3. Excretion, of some foreign chemicals, such as drugs, pesticides, and food additives, and their metabolites.
4. Gluconeogenesis. During prolonged fasting, the kidneys synthesize glucose from amino acids and other precursors and release it into the blood. The kidneys can supply approximately 20 percent as much glucose as the liver does at such times.
5. Kidneys act as endocrine glands, secreting some important hormones like erythropoietin, renin, and **1,25-dihydroxy vitamin D₃** and Prostaglandin synthesis. Also catabolism of polypeptide hormones (e.g., parathyroid hormone, insulin) occurs in the kidney (*Vander et al., 2001*).

Many renal functions are shared with other organs (acid-base control with lung; blood pressure control via the renin-angiotensin-aldosterone axis with liver, lung and adrenal glands). Other functions are not routinely measured