Renal Failure in Intensive Care

**Essay Submitted for Partial Fulfillment
of Master Degree in Intensive Care**

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تمت مناقشة الرسالة

بسم الله الرحمن الرحيم

قالوا سبحانك لا علم لنا

إلا ما علمتنا إنك أنت

العليم الحكيم

صدق الله العظيم

سورة البقرة - الآية ٣٢

بسم الله الرحمن الرحيم

To My Family

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Introduction

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Acute renal failure (ARF) is defined as acute (sudden), usually reversible deterioration of renal function with accumulation of waste products (urea, creatinine, potassium) and serious disorders in internal environment, often, but not always, associated with a reduction in the daily urine output (less than 400 ml per day) and in some cases there may be total anuria ***(Taube, 1996; and El-Said, 1992)***, i.e. oliguria is not a required diagnostic criterion ***(Mather, 1989)*** and may not be due to ARF, e.g. excess antidiuretic hormone (ADH).

Oliguric renal failure should be accompanied by an increase in serum creatinine of at least 0.5 mg/dl above baseline or 50% over baseline, a reduction in calculated creatinine clearance of at least 50%, or severe renal dysfunction requiring some form of renal replacement therapy ***(Minos, 1998)***.

In the intensive care unit (ICU), patients with renal failure are usually in the setting of multiple organ failure (MOF) rather than single organ (renal) failure, which is usually managed outside the ICU.

Incidence:

Approximately 15% of patients admitted to general ICU will have or will develop ARF ***(Trann et al., 1990)*** although the incidence in certain predisposed groups of critically ill patients (e.g., those with sepsis or liver failure, or with cardiovascular insufficiency) may be much higher ***(Taube, 1996)***. However, the relative incidence of ARF in a large multi-disciplinary ICU subserving complex cardiothoracic, vascular and liver transplant surgery will be much higher *(Taube, 1996)*.

The pattern of ARF is also constantly changing. Thus, improvements in cardiac surgery and obstetrics have reduced the incidence of ARF in these settings, only for new high-risk procedures such as liver and cardiac transplantations to emerge. Acute renal failure in pregnancy is rare although still associated with a high mortality ***(Turney et al., 1989)***.

Acute renal failure can have a mortality of 80% in critically ill patients and the introduction of acute hemodialysis has not reduced the mortality in ARF, which has serious implications ***(Anderson, 1993; and Thadani et al., 1996)***.

Causes and Pathogenesis of Renal Failure in ICU

Causes and Pathogenesis of Renal Failure in ICU

The standard robust classification of pre-renal, intrinsic renal, and post-renal is still very useful and clinically effective ***(Taube, 1996)***. Pre-renal ARF is due to reduction in renal blood flow (RBF) and the development of acute tubular necrosis (ATN). Intrinsic renal failure encompasses the various forms of rapidly progressive glomerulonephritis (RPGN) and acute tubulo-interstitial nephritis (ATIN); whereas post-renal refers to ARF caused by obstruction of the urinary tract.

In a multi-disciplinary ICU, particularly in the setting of multiple organ failure, ARF is usually pre-renal. However, not all causes fit comfortably into this classification. ARF at the bedside is usually multifactorial, often complicated, if not caused, by sepsis, hypovolemia and drugs, and the etiology of a particular patient renal failure may therefore change during the course of their ICU stay.

Moreover, many patients with ARF in the ICU have preexisting renal disease, or are old and chronically ill and the initiating cause may not be the only cause, particularly if patients have a prolonged ICU stay. Although ARF is readily treatable with dialysis and hemofiltration and theoretically reversible, the prognosis of these patients is poor with an overall mortality of approximately 80% ***(Taube, 1996)***.

Causes of Pre-Renal ARF:

The pre-renal sources of ARF are located proximal to the kidneys and are responsible for roughly half the cases of ARF and are characterized by a decrease in renovascular flow ***(Anderson, 1993; Garella, 1993; and Thadani et al., 1996)*** **(table 1)**.

The autoregulation of renal blood flow by vasodilatory prostaglandins and nitric oxide protects the kidney during hemorrhage despite systemic vasoconstriction ***(Vatner, 1974)*** and low blood pressure. Being generally recognized and treated, hypovolemia is now an increasingly rare sole cause of pre-renal ARF. Similarly, established pre-renal ARF due to low cardiac output states associated with myocardial infarction, cardiac surgery, tamponade, valvular dysfunction or cardiomyopathy is relatively rare in clinical practice. This is because the cause of low cardiac output is either rapidly reversed or the patient dies with intractable heart failure before established renal impairment becomes a clinical problem ***(Taube, 1996)***.

Sepsis has been found to precede ARF in up to 50% of patients in the ICU ***(Trann et al., 1990; and Groeneveld et al., 1991)***. Despite systemic vasodilatation, renal blood flow is reduced during endotoxemia in animals ***(Lugon et al., 1989; O'Hain et al., 1989; and Shaer et al., 1990)*** as well as in sepsis in humans ***(Tristan and Cohn, 1970; and Brenner et al., 1990)***. Intra-renal blood flow is altered, with cortical vasoconstriction and relative preservation of juxta-glomerular and medullary blood flow ***(Lambalgen et al., 1991)***.

Cytokines including tumor necrosis factor (TNF), interleukin-1 (IL-1), and platelet activating factor (PAF) are released in response to endotoxins. They affect renal hemodynamics, activate complement and leukocytes and damage vascular endothelium with intravascular coagulation as well as glomerular and intertubular capillary thrombosis. Endotoxins further reduce renal blood flow by promoting the synthesis and release of endothelins ***(Takahashi et al., 1990)*** and thromboxane A_2 ***(Badr et al., 1986)***.

Table (1): Causes of pre-renal ARF.

A. Low intravascular volume:

1. Hemorrhage: e.g., GIT, rupture spleen, accidental hemorrhage, placenta previa, abortion, crush syndrome.
2. Plasma losses: burns, acute pancreatitis.
3. Loss of fluids and electrolytes:
    a. GIT losses: diarrhea, prolonged vomiting, enterocutaneous fistula or ileostomy, intestinal obstruction, paralytic ileus.
    b. Renal losses: excessive use of diuretics, osmotic diuretics (hyperglycemia, and uncontrolled D.M.), "salt losing" nephropathy, hypoaldosteronism (Addison's disease).
    c. Skin losses: excessive sweating (heat stroke).
4. General anesthesia and surgical operations causing hypotension and/or blood loss.

B. Low effective intravascular volume:

- Low cardiac output states (acute heart failure, post-myocardial infarction and cardiac surgery, cardiomyopathies, tamponade).
- Ascites / cirrhosis (hepatic failure).
- Massive pulmonary embolism.
- Nephrotic syndrome
- Positive pressure ventilation.

C. Third-Space losses:

- Acute abdomen and peritonitis.
- Postoperative abdominal surgery.
- Hypoalbuminemia.
- Soft tissue trauma.
- Pancreatitis.

D. Renal ischemia with normal blood pressure:

- Renal artery and vein thrombosis, embolization, dissection
- Distal cholesterol embolization.

E. Hypercalcemia.

F. Sepsis: (gram negative, e.g., E. coli).

G. Drugs: e.g., noradrenaline and dopamine (high dose), ACEI, NSAIDs, cyclosporine.

(Taylor and Delinger, 1989; and El-Said, 1992 with modification)