Septic Acute Kidney Injury in critically ill patient

An Essay

Submitted for Partial Fullfillment of

Master Degree in the Intensive Care

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ADH	Antiduretic Hormone
AIFR	Adequate initial fluid resuscitation
AKI	acute kidney injury
ALI	Acute lung injury
APACHE	Acute Physiology and Chronic Health Evaluation scoring system
APC	activated protein C
ARDS	Acute respiratory distress syndrome
ARF	acute renal failure
AT	anti thrombin
ATP	adenosine tri phosphate
CECs	circulatory endothelial cells
CHFD	continuous high flux dialysis
cGMP	cyclic guanosine mono phosphate
CLFM	conservative late fluid management
CRP	C reactive protein
CRRT	continuous renal replacement therapy
CVC	Central venous catheter
CVP	Central venous pressure
DIC	Dissiminated intra vascular coagulopathy
EC	Endothelial cells

Table of appreviations

ET	Endothlins
FLC	Free light chain
GAGs	Glycosaminoglycans
GCP	Glomerular capillary pressure
GFR	Glomerular filtration rate
HCO	High- cut off
HS	Heparan sulfate
HVHF	High volume hemofiltration
IL	Interleukins
ILra	Interleukin receptor antagonist
I NOS	Inducible nitric oxide synthase
IVIG	Intravenous immunoglobulins
JGA	Juxta glomerular apparatus
LBP	Lipopolysacharide Binding Protien
LPS	Lipopolysacharide
MAP	Mean Arterial Pressure
MIF	Macrophage Migration Inhibitory Factor
MPs	Micro Particles
MR	Myogenic Response
NF-KB	Nuclear Factor-KB
NGAL	Neutrophil Gelatinase Associated Lipocain
NO	Nitric Oxide

PAC	Pulmonary Artery Catheter
PAF	Platelet Activating Factor
PAI-١	Plasmin Activator Inhibitor-١
PAOP	Pulmonary Artery Occlusion Pressure
PEEP	Possitive End Expiratory Pressure
PG	prostaglandins
PMN	Polymorph Nuclear Neutrophils
RBF	Renal Blood Flow
ROS	Reactive Oxygen Spices
SAFE	Saline versus Albumin Fluid Evaluation
SCVO٢	Central Venous Oxy-haemoglobin Saturation
SIRS	Systemic Inflammatory Response Syndrome
SLED	Sustained Low Effeciency Dialysis
SOFA	Sequential Organ Failure Assessment
S'VO٢	Mixed venous Oxy haemoglobin Saturation
TGF	Tubulo Glomerular Feedback
TF	Tissue Factor
TFPI	tissue Factor Pathway Inhibitor
TLR	Toll –like Receptor
TM	Thrombomodulin
TNF	Tumor Necrosis Factor
TPA	Tissue Plasminogen

TREM-١	Triggering Receptor Expressed on Myeloid cells
TX	Thromboxane
uPA	Urokinase-type plasminogen activator

Acknowledgement

*First, thanks are all due to **Allah** for Blessing this work until it has reached its end, as a part of his generous help throughout our life.*

*I am deeply grateful to **Prof. Dr. Ibrahim Abd El-ghani Ibrahim,** Professor of Anesthesia and Intensive Care, Faculty of Medicine, Ain Shams University for sponsoring this work, and his keen supervision and without his support it was impossible for this study to be achieved in this form. I had the privilege to benefit from his great knowledge, and it is an honor to work under his guidance and supervision.*

*I am also greatly indebted to **Dr. Randa Ali Shoukry,** Assistant professor of Anesthesia and Intensive Care, Faculty of Medicine, Ain-Shams University, for her great supervision, great help, available advises, continuous encouragement.*

*I would like to direct my special thanks to **Dr. Mohammed Eldesouky Mohammed,** Lecturer of Anesthesia and Intensive Care, Faculty of Medicine, Ain Shams University, for his invaluable help, fruitful advice, continuous support.*

*I want also to thank **my family** for supporting me throughout my life.*

Amira Ramadan Amin

Introduction

Sepsis, a commonly encountered scenario in an intensive care unit (ICU), often leads to multi-organ dysfunction and the kidney is one of the organs frequently afflicted. Acute kidney injury (AKI) occurs in about ١٩٪ of patients with moderate sepsis, ٢٣% with severe sepsis and ٥١٪ with septic shock, when blood cultures are positive. (**frausto et al., ١٩٩٥**)

Septic AKI had a higher in-hospital mortality rate, compared with nonseptic AKI (٧٠,٢ vs. ٥١,٨٪) (Morimatsu H ;et al .,٢٠٠٧) This indicates that the mortality rates of acute kidney injury in septic critically ill patients remains high despite of our increasing ability to support vital organs. (**Uchino et al .,٢٠٠٥**)

The beginning and ending supportive therapy (BEST) kidney investigators inferred that septic AKI was associated with greater derangement in hemodynamic and laboratory parameters, greater severity of illness and higher need for mechanical ventilation and vasopressor therapy. A few more facts emerged from this study. Oliguria was found to be more common in septic AKI (٦٧ vs ٥٧٪) Median duration of ICU and hospital stay for survivors (٣٧ vs. ٢١d), was longer for septic AKI.(**Bagshaw etal., ٢٠٠٧**)

Distinguishing between septic and non-septic AKI, therefore, may not just be of academic interest but may have clinical relevance for physicians. It has been suggested that septic AKI may have a distinct pathophysiology as well.(**wan etal.,٢٠٠٨**)

Septic AKI may have a unique identity and responses to interventions and outcome may be different in this group of patients, when compared to those with non-septic AKI . Significant progress has been made, over the years, towards learning how to detect AKI early, agreeing on an international consensus definition, delineating the pathophysiologic mechanisms which predispose to a high incidence of AKI in sepsis, trying to deduce logical protective and preventive strategies and finally on how to deliver the optimal renal support when the kidney fails. (**Majumdar, ٢٠١٠**)

Aim of the work

The aim of this essay is to provide information about diagnosis and pathophysiology of septic acute kidney injury in ICU, also to focus on the update on the current state of intervention in septic acute kidney Injury.

Prevention, pharmacological support and extra-corporeal blood purification also will be reviewed and discussed.

Physiology of the kidney

Structure of the kidney

A kidney has an outer fibrous renal capsule and is supported by adipose tissue. It has two main parts **(figure ١)** :

● Outer cortex – this is reddish-brown and is the part where fluid is filtered from blood.

● Inner medulla – this is paler in colour and is made up of conical-shaped sections called renal pyramids. This is the area where some materials are selectively reabsorbed back into the bloodstream. (**Helen,** ٢٠١٠)

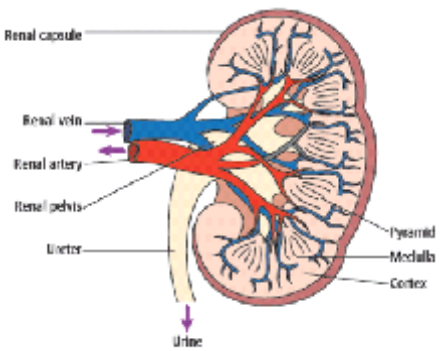

Figure (١):structure of the kidney***(Helen ,٢٠١٠)***

Physiological functions of the kidney

First,the 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.

Second, the kidneys 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.

A third function of the kidneys is the excretion, of some foreign chemicals, such as drugs, pesticides, and food additives, and their metabolites.

A fourth function is 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 ٢٠ percent as much glucose as the liver does at such times.

Finally, the kidneys act as endocrine glands, secreting some important hormones like erythropoietin, renin, and ١،٢٥-dihydroxyvitamin D٣ and Prostaglandin synthesis. Also catabolism of polypeptide hormones (e.g., parathyroid hormone, insulin) occurs in the kidney (**Vander et al.,**)

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 (small peptide excretion, tubular metabolism, hormonal production) in the ICU and are not considered clinically important. There are only two physiological functions that are routinely and easily measured in the ICU, which are unique to the kidney and which are considered clinically important: the production of urine and the excretion of water soluble waste products of metabolism. Thus, clinicians have focused on these two aspects of renal function to help them define the presence of acute renal failure (**Bellomo et al.,** ٢٠٠٤)