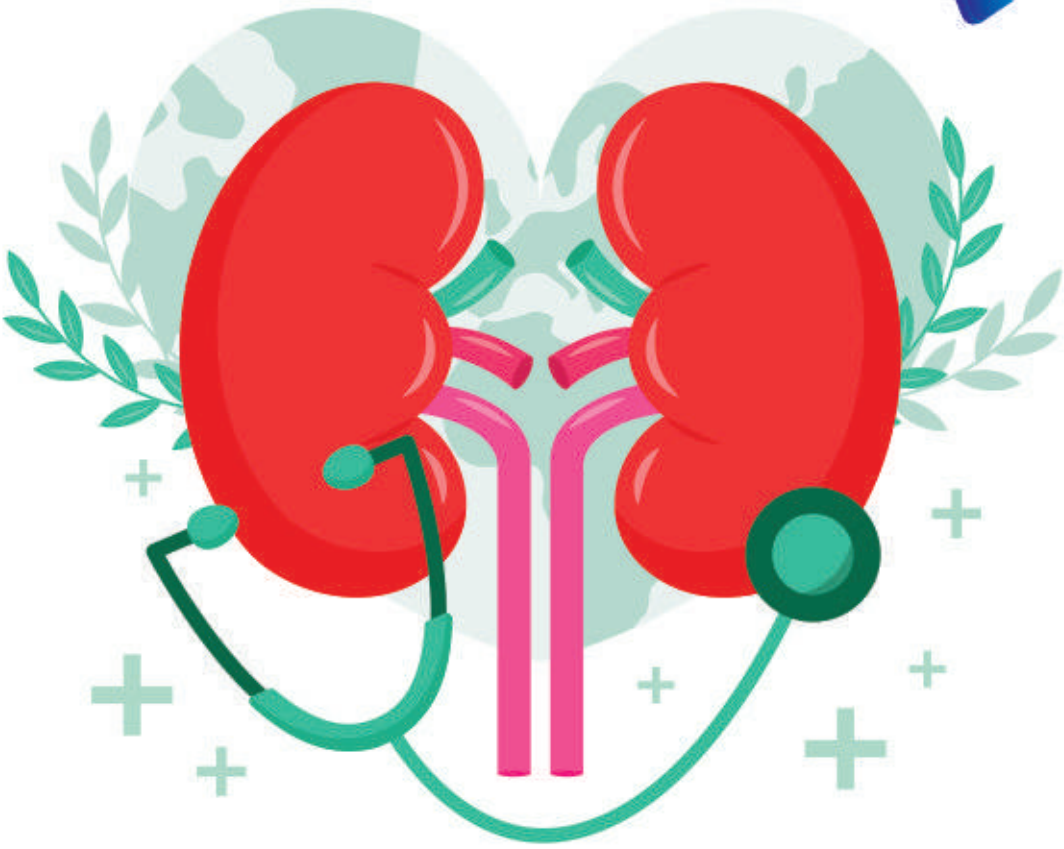
Arif Rohman Mansur | Yelly Herien



RENAL RESILIENCE

STRATEGIES FOR MANAGING ACUTE KIDNEY INJURY

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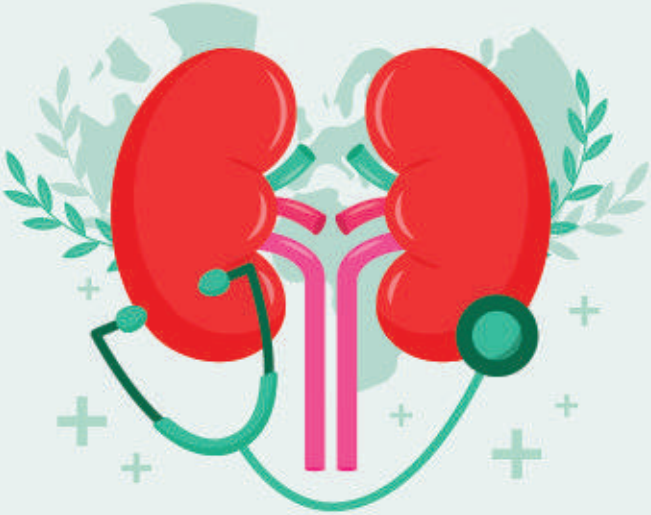

This book provides a comprehensive understanding of acute kidney failure (ARF), also known as acute kidney injury (AKI). In this book, you will find a clear introduction and definition of AKI, as well as frameworks such as the Acute Kidney Injury Network (AKIN), RIFLE Criteria, and Kidney Disease: Improving Global Outcomes (KDIGO) that are used for the diagnosis and classification of AKI. The epidemiology of AKI and its impact on neonates is also discussed. The etiology of AKI is described in depth, including prerenal injury, intrinsic renal disease, and postrenal causes associated with urinary tract obstruction. The pathophysiology of AKI and its developmental phases are also described in detail, including the causative factors of prerenal, intrarenal, and postrenal AKI.

Prevention of AKI is the next concern, with an emphasis on surveillance, maintenance of health, and reduction of modifiable risk factors. This book also describes various diagnostic studies, including blood tests, urine tests, and other diagnostic tests useful in diagnosing AKI. Management of AKI is the next focus, with discussions on medical management, pharmacological therapy, diet management, peritoneal dialysis and hemodialysis procedures. This book also provides insight into the prognosis of AKI. This book also gives special attention to quality nursing management in the care of AKI patients. Nursing assessment, physiological and psychosocial evaluation, nursing diagnosis, and planning and goals of comprehensive nursing care are also described. nursing Intervention.

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Yelly Herien

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PREFACE

This book is a comprehensive guide providing in-depth knowledge of acute kidney injury (AKI) and its related aspects. AKI is a serious condition that can be life threatening and requires good understanding and proper treatment. In this book, we have compiled structured and informative content to provide a complete knowledge of AKI. Starting from an introduction to AKI, clear definitions, to frameworks such as the Acute Kidney Injury Network (AKIN), RIFLE Criteria, Kidney Disease: Improving Global Outcomes (KDIGO) which are used in the diagnosis and classification of AKI. We also discuss epidemiology, including the categories of AKI and their impact on neonates.

In the etiology section, we examine the causes of AKI, such as prerenal injury, intrinsic kidney disease, and postrenal causes associated with urinary tract obstruction. We also describe in detail the pathophysiology of AKI, including the causative factors of prerenal, intrarenal, and postrenal AKI, as well as the phases that occur in the development of AKI.

The next section addresses the prevention of AKI, with an emphasis on monitoring, health maintenance, and efforts to reduce modifiable risk factors. We also describe various diagnostic studies, including blood tests, urine tests, and other diagnostic tests that help in the diagnosis of AKI.

The management of AKI is the focus of subsequent chapters, where we discuss medical management, pharmacological therapy, dietary management, as well as peritoneal dialysis and hemodialysis procedures that may be required in more complex cases. We also provide insight into the prognosis of AKI.

Not to forget, this book also pays attention to the importance of good nursing management in the care of AKI patients. We cover nursing assessment, physiological and psychosocial evaluation, nursing diagnosis, and comprehensive nursing care planning and goals. Nursing interventions, such as providing drug therapy, meeting nutritional needs, preventing infection, and emotional support, are also our concern in this book.

Finally, we present a brief summary, conclusion, and practice questions as a form of reader enrichment. A detailed bibliography is also included to enable the reader to continue exploring for more in-depth information. The glossary at the end of the book will help readers understand terms specifically used in the context of AKI.

We hope that this book can provide a meaningful contribution to medical professionals, students, and all parties interested in deepening their knowledge of AKI.

Warm regards,

Arif Rohman Mansur

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Arif Rohman Mansur
Yelly Herien

CHAPTER 1

ACUTE KIDNEY INJURY

A. Introduction

The term acute kidney failure (AKI) was first introduced by Homer W. Smith in 1951, but until 2004 there was no consensus regarding the diagnostic criteria for AKI. require renal replacement therapy (RRT). AKI is not only ARF, but refers to a collection of signs of changes in kidney function that are minimal to changes that require dialysis therapy(Pardede & Puspaningtyas, 2012).

Figure 1. Homer W. Smith
Source:https://www.kidneynews.org/view/journals/kidney-news/7/10/11/article-p28_22.xml

CHAPTER 2

ETIOLOGY OF ACUTE KIDNEY INJURY

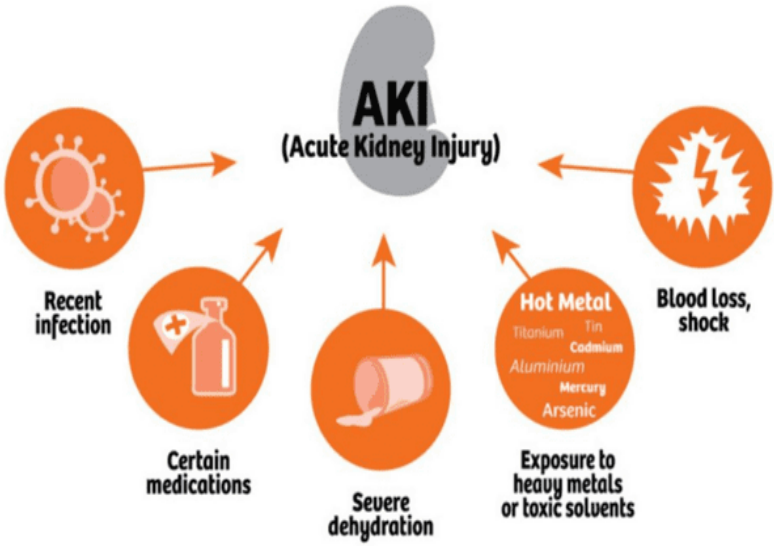

Figure 9. Etiology Acute Kidney Injury
Source:*https://lakhanikidneycare.com/acute-kidney-injury/*

One of the most important functions of the kidneys is the filtering and excretion of nitrogenous waste products from the blood. Measurements of increased blood urea nitrogen (BUN) and creatinine serve as indicators of decreased kidney function indicating decreased clearance of these waste products. AKI is currently defined as a rapid decrease in the glomerular filtration rate (GFR) resulting in retention of nitrogenous wastes, especially creatinine and blood urea nitrogen. Consequently, current diagnosis relies on serial measurements over time of these substances in the

CHAPTER 3 PATHOPHYSIOLOGY OF ACUTE KIDNEY INJURY

The causes of acute kidney failure (AKI) can be divided into three categories, namely prerenal, intrarenal, and postrenal. Prerenal AKI is the most common type, accounting for about 55% of the total cases. In prerenal AKI, hypoperfusion occurs which results in damage to the kidney without directly affecting the integrity of the kidney tissue itself. Whereas intrarenal AKI, also known as intrinsic AKI, occurs as a result of direct damage to functional kidney tissue, and is responsible for the other 40% of cases. Postrenal AKI, which is the rarest type, results from obstruction of the urinary tract causing damage to the kidneys(Nair & Peate, 2013).

A. Prerenal Causes

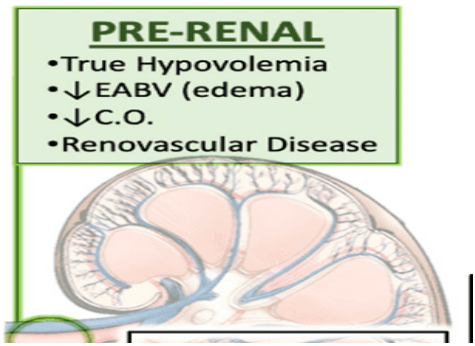

Figure 10. Pre Renals

Source: https://www.grepmed.com/images/12208/acute-causes-injury-aki-differential

CHAPTER 4

PREVENTION OF AKI

The following steps should be taken to prevent AKI in high risk groups/scenarios:

A. Monitor (Observation)

Children should have their creatinine checked and repeated if there is concern. The child's fluid balance including urine output, weight, urinalysis and Pediatric Early Warning Score (PEWS) should also be recorded and reviewed daily. Any signs of sepsis should be checked and treated immediately.

B. Maintenance

Attention must be paid to the child's circulating volume to ensure that the child has adequate circulating volume and perfusion pressure. Hypo-perfusion must be treated promptly with fluid boluses and inotropic support once the child's volume is full.

C. Minimize

Further damage should be reduced by reviewing, adjusting and monitoring drugs that may affect kidney function e.g. NSAIDs, ACEIs, ARBs, aminoglycosides ***and calcineurin inhibitors***. Intravenous contrast should also be avoided if possible(Kidneys, 2019).

D. Target modifiable risk factors

Targeting modifiable risk factors is an important strategy in efforts to reduce the risk of acute kidney injury (CS-AKI). Some modifiable risk factors include avoiding poor renal

CHAPTER 5 DIAGNOSTIC STUDIES

Assessment and diagnosis of patients with Acute Kidney Injury includes evaluation of changes in urine, diagnostic tests that evaluate the contours of the kidneys, and variations in normal laboratory values.

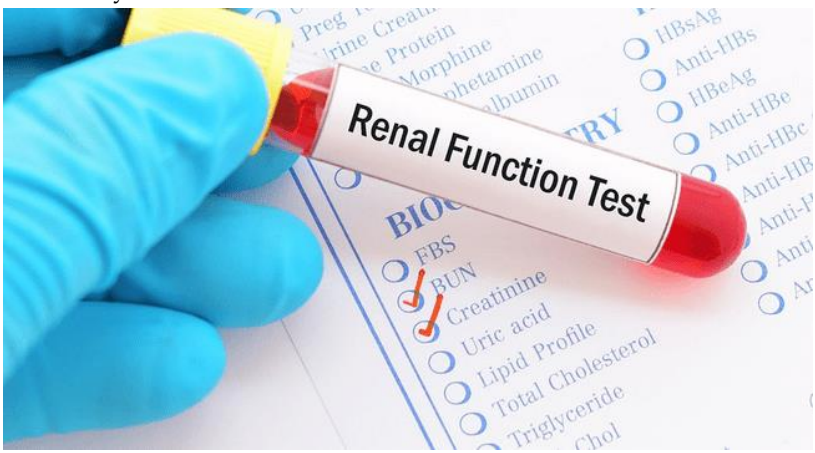

Figure 15. Renal Function test
Source:https://potreromed.com/acute-kidney-injury-signs-symptoms-and-criteria/

A. Blood Tests

Blood urea nitrogen (BUN): Measures the waste product of protein metabolism in the liver, is filtered by the kidneys and eliminated in the urine. (Elevated BUN levels strongly suggest kidney dysfunction, although liver disorders, hydration status, and other factors can also affect BUN levels).

CHAPTER 6 MANAGEMENT OF AKI

A. Medical management

The goal of treatment is to restore normal chemical balance and prevent complications until repair of kidney tissue and restoration of kidney function can occur. General management principles for acute kidney injury include determination of volume status, fluid resuscitation with isotonic crystalloids, treatment of volume excess with diuretics, discontinuation of nephrotoxic drugs, and adjustment of drug prescription according to renal function.(Mercado et al., 2019).

Pharmacological Therapy. Cation exchange resins or Kayexalate can reduce elevated potassium levels; IV dextrose 50%, insulin, and calcium replacement can be given to move potassium back into the cells; Diuretic agents are often given to control fluid volume.

Nutrition Therapy-Dietary protein replacement is individualized to provide maximum benefit and minimize uremic symptoms; similarly, caloric needs are met with high-carbohydrate foods, since carbohydrates have a protein-sparing effect; limited foods and liquids containing potassium or phosphorus; and after the diuretic phase, the patient was put on a high protein, high diet.

CHAPTER 7 NURSING MANAGEMENT

Nurses have an important role in caring for patients with Acute Kidney Injury (AKI)

A. Nursing Assessment

PHYSIOLOGICAL EVALUATION

Check vital signs, level of consciousness, and other neurological indicators to help identify clinical signs of electrolyte imbalance. Weigh the child's weight on admission as a basis for evaluating changes in fluid status. Monitor urinalysis, urine culture, and blood chemistry studies. Check the color, specific gravity, amount, and smell of the urine. Cloudy urine may indicate infection, while tea-colored urine may indicate hematuria. Observe the specific gravity of the urine and the amount and frequency of fluid in and out.

PSYCHOSOCIAL EVALUATION

Unexpected and sudden hospitalization of children can cause anxiety for parents and children. Assess feelings of anger, guilt, or fear associated with hospitalization. This also applies to the feelings one might have if a child develops acute kidney failure as a result of dehydration, preventable injury, or poisoning. Review coping mechanisms, family support systems, and stress levels(Ball et al., 2017).

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GLOSSARY

Creatinine

Creatinine is a chemical compound that is formed as a by-product of creatine metabolism in muscles. Creatinine is a parameter commonly used in blood tests to evaluate kidney function. High concentrations of creatinine in the blood can indicate impaired kidney function or kidney damage. Creatinine is formed as a result of muscle creatine dehydration and is a waste product of creatine. Creatinine is filtered by the glomerulus of the kidney and is not reabsorbed by the tubules under normal conditions. Serum creatinine and creatinine clearance provide an overview of glomerular filtration. Clinical implications: Creatinine measurement is obtained from 24-hour urine collection, but it is difficult to perform.

Urinary Creatinine (Clcr) Creatinine clearance
Normal value
Man : 1 - 2 g/24 hours
Woman: 0.8 - 1.8 g/24 hours

Acute Tubular Necrosis (ANT): Structural injury or tissue necrosis in the kidney, caused by ischemia or toxic injury. Necrosis is usually fragmentary, but injury can be widespread. NTA should be suspected in individuals who present after experiencing hypotension due to cardiac arrest, bleeding, sepsis, drug overdose, or surgery.

Anuria: Urine production less than 100 mL/day.

Azotemia: Accumulation of nitrogenous waste products, especially urea, in the blood.

Calculus: Masses of solid material or metabolic substances—kidney or bladder stones.

Catabolic: Degrading metabolism or the breakdown of protein for

energy leads to muscle wasting, loss of healthy muscle mass, and a negative nitrogen balance.

Glomerular filtration rate (GFR): is a measure used to evaluate the level of blood filtration by the glomerulus, the main filtering unit in the kidney. GFR measures how efficiently the kidneys remove waste products and toxins from the blood and regulates the balance of fluids and electrolytes in the body. GFR is usually measured in volume per unit time (eg, mL/min). Normal GFR levels vary depending on a number of factors

Glomerulonephritis: Inflammation of the glomerular capillary walls, causing decreased filtration.

Hydronephrosis: Enlargement of the kidneys caused by urine flowing back from the bladder into the kidneys or the inability of urine to flow out of the kidneys into the bladder; excessive backflow stretches the kidney, causing functional damage to the kidney.

Myoglobin: The form of hemoglobin found in muscle tissue and released into the urine when tissue damage occurs.
Nephrotoxins: Chemicals, including drugs, that can cause kidney damage.

Nonoliguric AKI: Urine production of more than 400 mL/day.

Oliguria: Urine production less than 400 mL/day.

Orthostatic hypotension:A drop in blood pressure when a person stands up from a sitting or lying position; often associated with hypovolemia.

Renal Parenchymal Diseases: Damage and scarring of the connective tissue of the kidneys.

Polyuria: Passage of large amounts of urine (2 to 6 L/24 hours),

without concentration and regulation of waste products; occurs during the diuretic phase of AKI in head injury (diabetes insipidus [DI]), and diabetic ketoacidosis (DKA).

Porphyrin: The nitrogen-containing chemical component of hemoglobin.

Pyelonephritis: Infection of the renal marrow or renal cortex.

Renal Replacement Therapy (RRT): General term used for life-saving treatments in kidney failure, including hemodialysis, peritoneal dialysis, hemofiltration, and kidney transplantation.

Uremia: Toxic clinical syndrome associated with fluid, electrolyte and hormonal imbalances and metabolic abnormalities due to decreased kidney function(Doenges et al., 2014)

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The author was born inJepara on August 28, 1987 and is the fifth child of five siblings. He completed his primary education at Jambu IX Mlonggo Public Elementary School in 1999, junior secondary education at Jepara 1st Public Middle School in 2002 and Senior High School at Jepara 1 Public High School in 2005. The author holds a bachelor's degree (S.Kep) and education Profession Nurse (Ns) from the Nursing Science Study Program (PSIK) Faculty of Medicine, Gadjah Mada University (UGM) in 2009 and 2010. The author has worked at STIKes Madani Yogyakarta and has also served as Chair of the Nursing Science Study Program, Chair of the Research and Community Service Institute / LPPM, and Vice Chair 1 for Academic Affairs. Currently the author works as a Lecturer in the Child and Maternity Section, Faculty of Nursing, Andalas University from April 1 2019 until now. Apart from being a lecturer, he is also active in writing books, articles in mass media and journals, managing community service journals (Warta Pengabdian Andalas), Chair of The Quality Assurance Bachelor of Nursing Study Program, Unand Book Task Force Team. One of the writer's mottos is "Dare to Try and Keep Learning". The author has also written several books or learning modules and published several research results in national and international journals. The writer is married and has three sons. One of the writer's mottos is "Dare to Try and Keep Learning". The author has also written several books or learning modules and published several research results in national and international journals. The writer is married and has three sons. One of the writer's mottos is "Dare to Try and Keep Learning". The author has also written several books or learning modules and published several research results in national and international journals. The writer is married and has three sons.

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