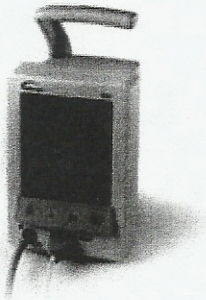
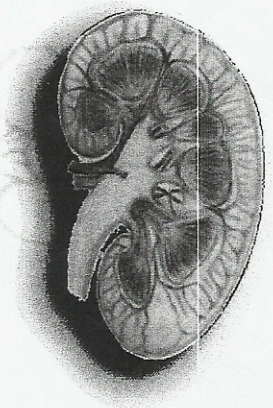
King Saud University
College of Medicine
Medical Education Department

RENAL BLOCK

CASE NO. (1)





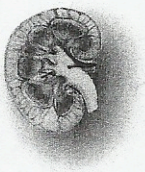
" In the operating room…"

Scenario:

Saleh, a 65 years old Saudi male is hypertensive and diabetic for 6 years. He has Osteoarthritis of the knees and was admitted for right knee replacement surgery. His medication includes Ibuprofen one tablet every 8 hours regularly, Lisinopril 20 mg PO OD, and Mixed Insulin (70/30) 20 units A.M. Subcutaneous and 8 units P.M.

His intra operative course was complicated by bleeding and drops in blood pressure and required blood transfusion. Post operatively his urine output has decreased significantly, the orthopedic surgeon decided to consult nephrology service.

NPO



On examination:

Saleh is not in distress. His vitals are as follows:

Vitals	Khalid's	Normal Range
Blood pressure	90/55 mmHg	130/80 mmHg
Pulse	116/min	60-100/min
Temperature	37.0 °C	36.6-37.2 °C
Respiratory rate	15/min	12-16/min

Jugular venous pressure is low

Cardiovascular examination:

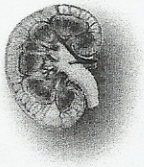
Normal first and second heart sound no added sound or murmurs.

Respiratory system examination:

Lungs are clear to auscultation and percussion.

Abdominal examination,

No tenderness, liver and spleen were not palpable.

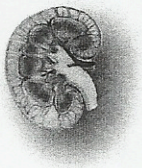


Investigation:

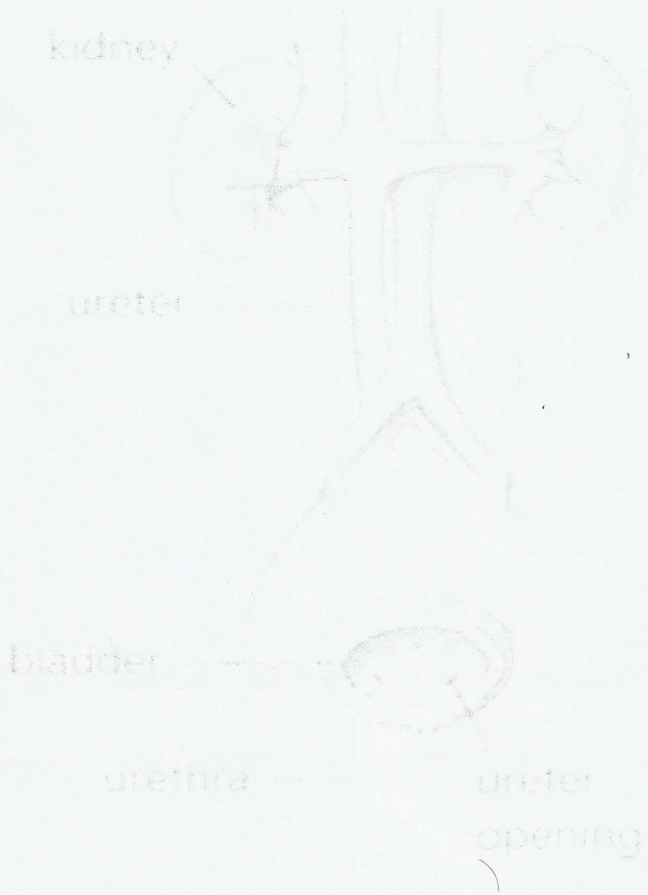
- Urine analysis: PH= 6, Protein +2, Hemoglobin +1,
  WBC= 12/HPF RBC=2 /HPF Granular cast is seen
  Specific gravity= 1.003
- Creatinine is 350 µmol/l (pre operative creatinine is 98 µmol/l)
- Urea 29 (pre operative level 3.5) K 6.2 mmol/l Na 138 mmol/l
- Complete blood count:
- WBC 5.6 Hb 70 Platelet 198

Ultrasound Kidneys:

- An ultrasound of the kidneys revealed bilateral kidneys of normal size with no obstruction.



The patient was diagnosed as having acute kidney injury (also known as acute renal failure), due to acute blood loss and hypotension causing acute tubular necrosis.



SECOND SESSION

Management:

Principles:

- Identify the etiology based on anatomical classification, pre renal, renal and post renal
- Depending on the etiology:
    - Pre renal: optimize volume status (if fluid loss replace, if blood loss transfuse, if congestive heart failure treat)
    - Post renal: relieve the obstruction
    - Renal : find out the etiology is it interstitial glomerular or acute tubular necrosis and treat accordingly
- Optimize volume status
- Avoid nephrotoxic medications including contrast, NSAIDs, Aminoglycosides
- Adjust medication dose according to renal function
- Renal replacement therapy is indicated if:
    - Volume over load refractory to medical treatment
    - Metabolic acidosis refractory to medical treatment
    - Hyperkalemia refractory to medical treatment
    - Uremic encephalopathy
    - Uremic pericarditis

Tutor Guide

Introduction:

It is a case of acute kidney injury (also known as acute renal failure), due to acute blood loss and hypotension causing acute tubular necrosis.

Learning Issues

1- Discuss the etiology based on the anatomical classification (pre-renal, renal and post-renal) for acute kidney injury.

2- Discuss the pathophysiology of acute kidney injury.

3- Discuss the pathophysiological change in the glomerular filtration rate in setting of acute kidney injury.

4- Discuss and recognize laboratory changes associated with acute kidney injury

5- Outline a management plan base on the pathophysiology discussed for acute kidney injury.

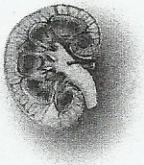
Distribute page1

Page 1

Saleh, a 65 years old Saudi male is hypertensive and diabetic for 6 years. He has Osteoarthritis of the knees and was admitted for right knee replacement surgery. His medication includes Ibuprofen one tablet every 8 hours regularly, Lisinopril 20 mg PO OD, and Mixed Insulin (70/30) 20 units A.M. Subcutaneous and 8 units P.M.

His intra operative course was complicated by bleeding and drops in blood pressure and required blood transfusion. Post operatively his urine output has decreased significantly, the orthopedic surgeon decided to consult nephrology service.

The students should start to ask questions about what has happened with the patient. They will discuss the different causes of such presentation. What evoked these symptoms? These should trigger some questions about what they would want to know in terms of a focused history. At an appropriate point in the discussion, when they need more information from the history and physical examination.



Distribute page2

On examination:

Khalid is not in distress. His vitals are as follows:

Vitals	Saleh's	Normal Range
Blood pressure	90/55 mmHg	130/80mmHg
Pulse	116/min	60-100/min
Temperature	37.0 °C	36.6-37.2°C
Respiratory rate	15/min	12-16/min

Jugular venous pressure is low

Cardiovascular examination:

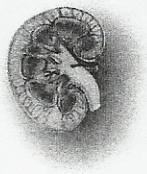
Normal first and second heart sound no added sound or murmurs.

Respiratory system examination:

Lungs are clear to auscultation and percussion.

Abdominal examination,

No tenderness, liver and spleen were not palpable.



Distribute page 3

Investigation:

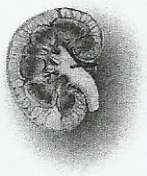
- Urine analysis: PH= 6, Protein +2, Hemoglobin +1,
  WBC= 12/HPF RBC=2 /HPF Granular cast is seen
  Specific gravity= 1.003
- Creatinine is 350 µmol/l (pre operative creatinine is 98 µmol/l)
- Urea 29 (pre operative level 3.5) K 6.2 mmol/l Na 138 mmol/l
- Complete blood count:
- WBC 5.6 Hb 70 Platelet 198
- An ultrasound analysis revealed bilateral kidneys of normal size with no obstruction.

At this point, the students should try to interpret the results and relevance of the investigations done in relation to the history and clinical examination and try to discuss differential diagnosis according to the new scope. The interpretation of these data should trigger learning issues in the students about physiology of kidney function, kidney functions tests,

Distribute page 4

The patient was diagnosed as having acute kidney injury (also known as acute renal failure), due to acute blood loss and hypotension causing acute tubular necrosis.

At this point, the students should ask about what causes acute kidney injury, risk factors, diagnosis, and management. The tutor must assure that the student's learning issues are in the line of the session objectives.



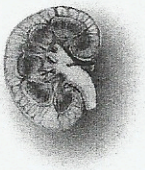
Second session

Distribute page 5

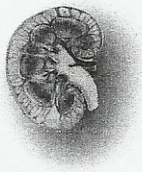
Management:

Principles:

- Identify the etiology based on anatomical classification, pre renal, renal and post renal
- Depending on the etiology:
    - Pre renal: optimize volume status (if fluid loss replace, if blood loss transfuse, if congestive heart failure treat)
    - Post renal: relieve the obstruction
    - Renal : find out the etiology is it interstitial glomerular or acute tubular necrosis and treat accordingly
- Optimize volume status
- Avoid nephrotixic medications including contrast, NSAIDs, Aminoglycosides
- Adjust medication dose according to renal function
- Renal replacement therapy is indicated if:
    - Volume over load refractory to medical treatment
    - Metabolic acidosis refractory to medical treatment
    - Hyperkalemia refractory to medical treatment
    - Uremic encephalopathy
    - Uremic pericarditis



In the second session, after the student feedback about lines of management, the tutor should distribute this page to criticize the case management and to conclude finally if they need any further information.



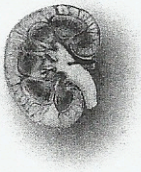
Reading material:

Acute kidney injury

Acute kidney injury (**AKI**), Also known previously as **acute renal failure** (**ARF**). AKI is has been generally defined as abrupt loss of kidney function that resulted in accumulation of nitrogenous waste product and dysregulation of fluid and electrolytes.

- What are the normal kidney functions?
    - Fluid Balance
    - Electrolyte Control
    - Acid-base balance
    - Metabolism and Excretion
    - Hormone production (Erythropoietin production, Renine ……)
- What abnormalities in function take place in setting of Acute Kidney Injury? How there are different from Chronic Kidney disease (CKD)?

AKI is diagnosed on the basis of clinical history, such as decreased urine production, and characteristic laboratory findings, such as elevated blood urea nitrogen and creatinine. Depending on its severity, AKI may lead to a number of complications, including metabolic acidosis, high potassium levels, changes in body fluid balance, and effects to other organ



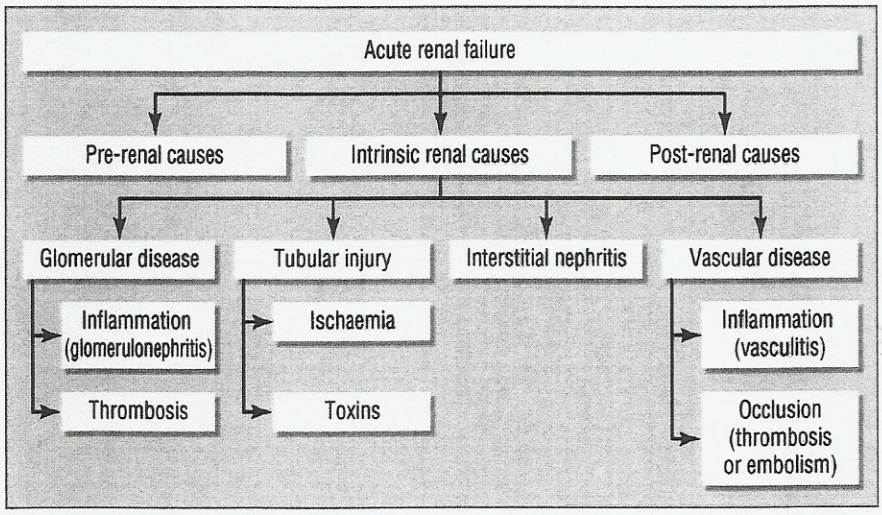
systems. Management includes supportive care, such as renal replacement therapy, as well as treatment of the underlying disorder.

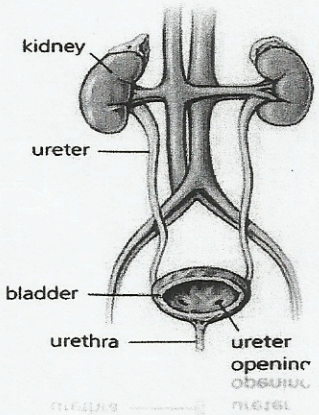
Epidemiology

- It occurs in
  - 5%of all hospitalized patients and
  - 35% of those in intensive care units
- Mortality is high:

Etiology of AKI:

The causes of acute kidney injury are commonly categorised into *prerenal*, *intrinsic*, and *postrenal*.





Prerenal

Prerenal causes of AKI are those that decrease effective blood flow to the kidney. These include systemic causes, such as low blood volume, low blood pressure, and heart failure, as well as local changes to the blood vessels supplying the kidney. The latter include renal artery stenosis,

which is a narrowing of the renal artery that supplies the kidney, and renal vein thrombosis, which is the formation of a blood clot in the renal vein that drains blood from the kidney.

- **Volume depletion**
  - Renal losses (diuretics, polyuria)
  - GI losses (vomiting, diarrhea)
  - Cutaneous losses (burns, Stevens-Johnson syndrome)
  - Hemorrhage
  - Pancreatitis
- **Decreased cardiac output**
  - Heart failure
  - Pulmonary embolus
  - Acute myocardial infarction
  - Severe valvular heart disease
  - Abdominal compartment syndrome (tense ascites)

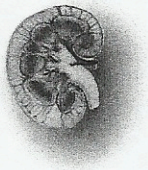
Intrinsic (Renal)

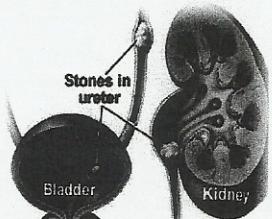
Sources of damage to the kidney itself are dubbed *intrinsic*. Intrinsic AKI can be due to damage to the glomeruli, renal tubules, or interstitium. Common causes of each are glomerulonephritis, acute tubular necrosis (ATN), and acute interstitial nephritis (AIN), respectively.

- **Glomerular**
  - Anti–glomerular basement membrane (GBM) disease (Goodpasture syndrome)
  - Anti–neutrophil cytoplasmic antibody-associated glomerulonephritis (ANCA-associated GN) (Wegener granulomatosis, Churg-Strauss syndrome, microscopic polyangiitis)
  - Immune complex GN (lupus, postinfectious, cryoglobulinemia, primary membranoproliferative glomerulonephritis)
- **Tubular**
  - Ischemi
  - Totoxic
    - Heme pigment (rhabdomyolysis, intravascular hemolysis)
    - Crystals (tumor lysis syndrome, seizures, ethylene glycol poisoning, megadose vitamin C, acyclovir, indinavir, methotrexate)
    - Drugs (aminoglycosides, lithium, amphotericin B, pentamidine, cisplatin, ifosfamide, radiocontrast agents)
- **Interstitial**
  - Drugs (penicillins, cephalosporins, NSAIDs, proton-pump inhibitors, allopurinol, rifampin, indinavir, mesalamine, sulfonamides)
  - Infection (pyelonephritis, viral nephritides)
  - Systemic disease (Sjogren syndrome, sarcoid, lupus, lymphoma, leukemia, tubulonephritis, uveitis

Postrenal

Postrenal AKI is a consequence of urinary tract obstruction. This may be related to benign





prostatic hyperplasia, kidney stones, or an obstructed urinary catheter.

- **Ureteric obstruction**
  - Stone disease,
  - Tumor,
  - Fibrosis,
  - Ligation during pelvic surgery
- **Bladder neck obstruction**
  - Benign prostatic hypertrophy [BPH]
  - Cancer of the prostate
  - Neurogenic bladder
  - Drugs(Tricyclic antidepressants, ganglion blockers,
  - Bladder tumor,
  - Stone disease, hemorrhage/clot)
- **Urethral obstruction** (strictures, tumor)

Classic laboratory findings in AKI

Type	U_{Osm}	U_{Na}	Fe_{Na}	BUN/Cr
Prerenal	>500	<10	<1%	>20
Intrinsic	<350	>20	>2%	<15
Postrenal	<350	>40	>4%	>15

Diagnosis:

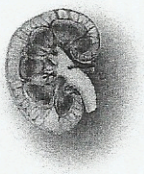
Acute kidney injury is diagnosed on the basis of clinical history and laboratory data. A diagnosis is made when there is rapid reduction in kidney function, as measured by serum creatinine, or based on a rapid reduction in urine output, termed oliguria.

GFR/Creatinine criteria	Urine Output criteria

Definition

Introduced by the Acute Kidney Injury Network (AKIN), specific criteria exist for the diagnosis of AKI:

1. Rapid time course (less than 48 hours)
2. Reduction of kidney function
    - Rise in serum creatinine
        - Absolute increase in serum creatinine of ≥0.3 mg/dl (≥26.4 μmol/l)
        - Percentage increase in serum creatinine of ≥50%
    - Reduction in urine output, defined as <0.5 ml/kg/hr for more than 6 hours



***R*isk**	Increase in creatinine x1.5 Or GFR decrease >25%	UO < .5ml/kg/hr for 6hrs
***I*njury**	Increase in creatinine x 2 Or GFR decrease >50%	UO < .5ml/kg/hr for 12hrs
***F*ailure**	Increase in creatinine x 3 Or GFR decrease >75%	UO < .3ml/kg/hr for 24 hrs or Anuria for 12hrs
***L*oss**	Persistent ARF = complete loss of renal function > 4 weeks	
ESRD	End Stage Renal Disease > 3 months	

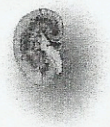
Staging

The *RIFLE criteria*, proposed by the Acute Dialysis Quality Initiative (ADQI) group, aid in the staging of patients with AKI:

Further testing

Once the diagnosis of AKI is made, further testing is often required to determine the underlying cause. These may include renal ultrasound and kidney biopsy. Indications for renal biopsy in the setting of AKI include:

1. Unexplained AKI
2. AKI in the presence of the nephritic syndrome
3. Systemic disease associated with AKI



Treatment

The management of AKI hinges on identification and treatment of the underlying cause.

Principle of management of AKI includes:

- Optimization of hemodynamic and volume status
- Avoidance of further renal insults (i.e. nephrotoxins. Such as NSAIDs and contrasts media)
- Optimization of nutrition
- If necessary, institution of renal replacement therapy.
- Frequent monitoring of renal function (serial serum creatinine) and urine output

Specific therapies

In prerenal AKI without fluid overload, administration of intravenous fluids is typically the first step to improve renal function. Volume status may be monitored with the use of a central venous catheter to avoid over- or under-replacement of fluid.

Should low blood pressure prove a persistent problem in the fluid-replete patient, inotropes such as norepinephrine and dobutamine may be given to improve cardiac output and hence renal perfusion. While a useful pressor, there is no evidence to suggest that dopamine is of any specific benefit and may be harmful.

The causes of intrinsic AKI require specific therapies. For example, intrinsic AKI due to Wegener's granulomatosis may respond to steroid medication. Toxin-induced prerenal AKI often responds to discontinuation of the offending agent, such as aminoglycoside, penicillin, NSAIDs, or acetaminophen.

If the cause is obstruction of the urinary tract, relief of the obstruction (with a nephrostomy or urinary catheter) may be necessary.

Diuretic agents

The use of diuretics such as furosemide, may be used to control fluid overload,

Renal replacement therapy

Renal replacement therapy, such as with hemodialysis, may be instituted in some cases of AKI.

Indication for renal replacement therapy:

- Symptoms of uremia (encephalopathy,…)
- Uremic pericarditis
- Refractory volume over load
- Refractory hyperkalemia
- Refractory metabolic acidosis

Complications

Metabolic acidosis, hyperkalemia, and pulmonary edemamay require medical treatment with sodium bicarbonate, antihyperkalemic measures, and diuretics.