ACUTE RENAL FAILURE

Acute Renal Failure Learning Objectives

At the completion of this module the student should be able to:

- Define GFR and be able to circulate it.
- Distinguish between a pre-renal, intrinsic renal, and post-renal causes of acute renal failure.
- Describe the pathogenesis and pathophysiology of these disorders.
- Describe the determinants of BUN and serum creatinine concentration.
- Describe the concept of fractional excretion of sodium and how it can be used to distinguish between pre-renal acute renal failure and acute tubular necrosis.
- Calculate the fractional excretion of sodium from urine and serum chemistries.
- Describe how urine creatinine to plasma creatinine ratio can help distinguish between pre-renal acute renal failure and acute tubular necrosis.
- Use urine chemistries, urine sediment, and clinical history to diagnose specific causes of acute renal failure.
- Describe the expected time courses for these disorders.
- Describe the role of afferent and efferent arteriolar vascular tone on glomerular filtration rate and the effect of prostaglandin and angiotensin converting enzymes inhibitors on afferent and efferent arteriolar vascular tone.
- List the biochemical and volume changes expected to result from acute renal failure.

Case 1:

A 43-year-old male is involved in an automobile accident. When paramedics arrive at the scene he is found to be in shock with a blood pressure of 60/40, a pulse of 120; his extremities are cold and clammy, and his abdomen is hard and tender. He is taken to the hospital receiving intravenous fluids during transport with his blood pressure increasing to 80/50 upon arrival to the ER. A Foley catheter is placed in the patient's bladder to monitor the urine output, which is scant. Upon arrival at the hospital a sample of this urine and a sample of blood are sent to the lab.

1) By the history alone, present a possible
   a. Pre-renal
   b. Intrinsic-renal
   c. Post-renal
   etiology for the patient's oliguria.

   Labs are obtained which show:
   BLOOD: Na = 140 mEq/l, K = 4.8 mEq/l, Cl = 102 mEq/l, $HC0_3$ = 24 mEq/l, BUN = 10 mg/dl (nl 5-15 mg/dl), Creat. = 1.0 mg/dl (nl 1.8-1.2 mg/dl),

   URINE: U_{Na} = 14 mEq/l, U_{creat} = 100 mg/dl.

2) If this patient is not making much urine and presumably is in renal failure, why are the BUN and Creatinine normal?

3) What factors detemine the rate that the BUN and creatinine rise when in renal failure?

4) Under what conditions might the creatinine rise disproportionately to the BUN, and visa-versa?

5) a. What does this patient's concentration of sodium in the urine (U_{Na}) tell you.
   b. What are the (renal) mechanisms by which the U_{Na} is lowered to this degree?
   c. What is a normal value of U_{Na}?

6) What is this patient's fractional excretion of sodium at this time (FE_{Na})?

7) This patient has had prolonged hypotension, could the patient have ischemic ATN as a cause of his oliguria?

Upon arrival at the hospital he is given several blood transfusions and he stabilizes. He receives a CT scan of his abdomen with intravenous contrast, which demonstrates a splenic rupture. He is taken to the operating room where he undergoes splenectomy. Concerned about infection, the surgeon orders gentamycin antibiotic which he receives before leaving the operating room. He is admitted to the surgical intensive care unit for his post-operative care. He is alert and oriented and his vital signs are now quite stable with a BP of 120/80, ° of 80, and a RR of 18. His cardiovascular exam is normal and he has no edema. His urine output however has not improved (12 ml/hr).

8) Again by the history alone, what are all the possible reasons at this point that the patient is oliguric?

Labs are obtained which show:
BLOOD: Na = 145 mEq/l, K = 5.6 mEq/l, Cl = 109 mEq/l, $HC0_3$ = 22 mEq/l, BUN = 30 mg/dl, Creat. = 1.9 mg/dl. The serum osmolality is 304 mosm/l.

URINE: A urinalysis shows a specific gravity of 1.010, has no blood or protein on dipstick, microscopic examination shows many coarse granular casts but is otherwise normal without RBC or WBC. URINE chemistries show a U_{Na} = 50 mEq/l, U_{creat} = 38 mg/dl, U_{osm} = 310 mosm/l.

9) Based on the recent lab values, what do you think is going on?

10) What is the ratio: U_{Creat}/P_{Creat} , a measure of and how might it help distinguish pre-renal causes of acute renal failure from ATN?

11) Why is the fractional excretion of sodium (FE_{Na}) the most useful of the urinary indices in evaluating patients with acute renal failure?

12) The fractional excretion of sodium (FE_{Na}) is the percent of filtered sodium that is excreted in the urine. Derive the equation for FE_{Na} knowing that the GFR = creatinine clearance = $\mathbf{U_{creat}\ V / P_{creat}}$, where **V**= urine flow rate.

13) What is the estimated GFR (creatinine clearance) if you know that the urine output is 12 ml/hr?

14) What are the mechanisms proposed for the reduction of GFR in ATN?

15) What might a renal biopsy show in ATN?

16) What would be the expected course of the patients urine output and renal function?

Case 2:

A 60-year-old male with a long history of diabetes and hypertension (but normal renal function) is admitted to the hospital with the acute onset of substernal chest pain. He is found to have had a myocardial infarction involving the entire anterior wall of the left ventricle. During the course of his hospitalization he is noted to gain 12 lbs., and develops peripheral edema. His urine output is only 400 ml/day. A chest X-ray shows pulmonary congestion and cardiomegaly.

On physical exam his BP is 100/60, P 90, RR 20. He is a mildly obese male in moderate respiratory distress. His neck demonstrated jugular venous distention. His lungs had rales at the bases bilaterally. The cardiac exam demonstrated a loud S_3 gallop. The PMI was displaced laterally. His extremities showed pitting edema. An echocardiogram demonstrated a poorly contracting left ventricle.

1) What type of ARF is suggested by the history and physical alone? Is it reversible?

Labs are obtained which show:
BLOOD: Na = 136 mEq/l, K = 5.6 mEq/l, Cl = 102 mEq/l, $HC0_3$ = 18 mEq/l, BUN = 100 mg/dl, Creat. = 3.0 mg/dl, Ca = 7.4 mg/dl (nl = 8.6-10.6), $P0_4$ = 8.2 mg/dl (nl=2.8-4.6).

URINE: S.G. = 1.030, pH = 5.0, no protein, no blood, no cells or casts. U_{Na} = 15 mEq/l, U_{Creat} = 160 mg/dl, U_{Osmol} = 550 mosm/l.

2) What is the FE_{Na}? Does that value confirm your clinical impression from above?

3) What other cardiac causes of ARF give a similar FE_{Na}?
What other non-cardiac causes of ARF give a similar FE_{Na}?

4) What would be the expected vascular tone of this patient's afferent and efferent arterioles?

5) What might be expected if a nonsteroidal anti- inflammatory agent (NSAIA) were given to this patient? Why doesn't this happen to someone with normal renal function?

6) Why is the BUN: creatinine ratio so high?
What is the expected course of the serum potassium, bicarbonate and phosphorous ?

7) What might a renal biopsy show?

Case 3:

A 70-year-old male presents to the emergency room because he stopped urinating. He has noticed difficulty starting and stopping his stream for the last six months. He states that the last time he was able to void (urinate) was about 72 hours ago. He also claims that he has abdominal pain and feels bloated. He has been healthy prior to this and he takes no medications.

On physical exam his BP was 140/90, ° 80, RR 20. The lungs were clear, the cardiac exam was normal, the abdominal exam showed a large mass that extended from his pelvis to his umbilicus. It was tender to palpation, and did not contain bowel sounds. His prostate was enlarged.

Labs are obtained which show:
BLOOD: Na = 138 mEq/l, K = 6.0 mEq/l, Cl = 108 mEq/l, $HC0_3$ = 16 mEq/l, BUN = 70 mg/dl, and Creat. = 5.6 mg/dl. URINE: No urine was available for analysis.

1) What is the likely diagnosis?

2) How could you confirm your clinical suspicion?

3) If a patient has only one kidney, how does this influence your differential diagnosis of ARF?

4) What other conditions might cause anuria?

5) What does the urine output tell you about the likelihood of obstruction in a patient?

6) If a patient has two kidneys and only one is obstructed, what would you expect the renal function to be?

7) Why do we always try to rule out obstruction in patients with renal failure?

8) What determines renal recoverability in patients with urinary obstruction?

9) Name two pathologic conditions that could cause obstruction at each segment of the kidney drainage system.
   a. renal pelvis , b. Ureters, c. Bladder, d. urethra