IV: Acute Renal Failure

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Specific Learning Objectives:

Knowledge

Subinterns should be able to describe and define:

1. The differential diagnosis of acute renal failure, including pre-renal, renal, and post renal causes

2. The utility of the urinalysis in the diagnosis of acute renal failure, including the significance of the following:

    a. Proteinuria

    b. Red or white cells casts

3. The utility of the urine sodium and fractional excretion of sodium in the differential diagnosis of acute renal failure

4. The utility of imaging modalities used in the evaluation of acute renal failure, including:

    a. Ultrasonography

    b. Renal scans

Skills

Subinterns should demonstrate the ability to:

1. Conduct a history eliciting and interpreting symptoms that could indicate uremia and fluid overload.

2. Conduct a physical examination detecting and interpreting signs of volume overload and bladder outlet obstruction

3. Develop a management plan for a patient with acute renal failure

    a. Create a differential diagnosis

    b. Perform and interpret a urinalysis

    c. Calculate a fractional excretion of sodium

    d. Use information from serum and urine laboratory studies and imaging studies to arrive at a specific cause

e. Communicate the expected course of acute renal failure and the procedure of hemodialysis to patients and families

f. Order appropriate fluid and electrolyte management, medication adjustment for decreased glomerular filtration rate, and supportive care

Attitudes and professional behaviors:
Subinterns should demonstrate:

1. Sensitivity to patients and families concerns and questions when confronted with a serious diagnosis such as acute renal failure

2. Commitment towards understanding patients' preferences regarding life-sustaining treatments such as dialysis

CASE: Acute Renal Failure

1) SCENARIO: A 71-year-old male with a history of hypertension presents to the Emergency Department with fever, chills, and dysuria. Two days ago he noticed dysuria and frequency of small amounts of urine. One day ago he experienced an onset of fever and a temperature of 102°, chills, vomiting (3-4 times/day), and flank pain (7/10). He started taking sodium naproxen for his flank pain with some relief (3/10) This morning the symptoms persisted and he had dizziness on standing, relieved by lying down. In the last year he had noticed hesitancy and nocturia times 2. He has a history of hypertension treated with Lisinopril 20 mg/day. His blood pressure has been under good control. His creatinine 2 months ago was 1.0 mg/dL. He has no other medical problems. In the Emergency Department his Blood Pressure was 80/50 mm Hg, Pulse was 100/min. Orthostatics were not done. He had no jugular venous distention supine. His lungs were clear. His heart had no murmurs or gallops. His abdomen had mild generalized tenderness and left flank was tender to percussion. He had no edema. His prostate was enlarged with no nodules.

2) ADDITIONAL DATA:
Labs revealed
Na 140 mEq/L K 6.2 mEq/L C1 102 mg/dL HCO_3 16 mEq/L
BUN 60 mg/dL Creatinine 2.4 mg/dLl
pH 7.32 on room air pO_2 90 mm Hg pCO_2 32 mm Hg HCO_3 16 mEq/L
Urinary Na 14 mEq/L
Urinary Cr 280 mg/dL
Urinalysis specific gravity 1.030 1+ protein microscopic 30-50 WBCs

1. Question: What is the differential diagnosis for his renal failure?
When patients present with renal failure it is either
Acute renal failure
Chronic renal failure
Acute renal failure with underlying chronic renal failure
With his creatinine two months ago being 1.0 mg/dL, this is most likely acute.
Possibilities for his acute renal failure:
Prerenal – His fractional excretion ($U_{Na} \times P_{Cr} / P_{Na} \times U_{Cr}$) x 100 is 0.1 %, which is consistent with prerenal
Reasons include:

- *Decreased po intake with vomiting*
- *Peripheral vasodilation from infection but with infection the kidney is constricted due to increased catecholamines and angiotensin II which further decreases renal blood flow*
- *NSAIDs worsening prerenal due to decreased renal prostaglandin production, which normally vasodilate the afferent artery. Without PG's there is unopposed vasoconstriction leading to decreased renal plasma flow thus worsening prerenal failure*
- *ACE inhibition decreasing angiotensin II causing decreased resistance in the efferent artery which decreases glomerular hydrostatic pressure worsening prerenal failure*
- *Hypotension decreasing glomerular pressure*

Renal

- *Pyelonephritis*
- *Acute tubular necrosis but fractional excretion of sodium is not consistent*

Post renal

- *Benign prostatic hypertrophy or prostate cancer since has enlarged prostate*

2. What radiological studies would you order?
Renal ultrasound looking for hydronephrosis and possible perinephric abscess
3. What are the reasons for increased potassium?
Answers:
Shift due to acidosis
Decreased renal function
ACE inhibition decreasing angiotensin II which causes a decrease in aldosterone
Rarely NSAIDs can cause hyporenin hypoaldosteronism

4. Question: What is acid base abnormality and mechanism of decreased bicarbonate?

Anion gap metabolic acidosis. Anion gap is 140 – (102 + 16) = 22. Using the Winter's equation for a metabolic acidosis the expected $pCO_2 = 1.5 (HCO_3) + 8 \pm 2$. Expected pCO_2 1.5 (16) + 8 ± 2 = 30 – 34. His pCO_2 is 32 so he has a compensatory respiratory alkalosis.

The kidney's two functions in acid base are to reabsorb bicarbonate and to replace the decrease in bicarbonate due to metabolism by secretion of titratable acid (mainly phosphate) and ammonium. With renal failure there is decreased ammoniogenesi as a result of decreased nephron mass.

5. Question: How would you treat the hypotension and the pyelonephritis?

Aggressive fluid resuscitation with normal saline initially 1-2 liters, along with antibiotics (quinolone or 3rd generation cephalosporin are usually given to cover likely gram negatives). If hypotension persists after fluid resuscitation consider norepenephrine to increase systemic vascular resistance or arginine vasopressin.

General Overviews:

ATN

Esson ML, Schrier RW. Diagnosis and treatment of acute renal failure. Ann Intern Med. 2002:137:744.