DR. CARR'S NOTES ON ACUTE RENAL FAILURE

Burn Team,

First, let's briefly talk about CHRONIC RENAL FAILURE. The National Kidney Foundation defines Chronic Kidney Disease as a decreased GFR of < 60mL/min/1.73m. There are 5 stages defining the degree of chronic kidney disease with stage 5 being Kidney Failure with a GFR < 15mL/min/1.73m.

Approximately 1 million nephrons are present in each kidney. With progressive loss of nephrons, the remaining healthy nephrons compensate by Hypertrophy & Hyperfiltration. Only after the GFR has decreased by 50% ('renal reserve') will there be an increase in plasma solutes such as urea and Cr.

The 2 leading causes of Chronic Renal Failure are DIABETES & HYPERTENSION. Independent predictors for chronic kidney disease are Diabetes, HTN, and Age.

Ultimately the Pathophysiology leading to progressively decreasing GFR is GLOMERULOSCLEROSIS with progressive loss of functional glomeruli and nephrons.

The 5 year survival rate for patients undergoing chronic dialysis is 35%. The life expectancy of a 60 year old starting hemodialysis is 4 years vs 20 years for a healthy 60 year old.

Patients with Chronic Kidney Disease generally don't manifest symptoms until the GFR < 30mL/min (Stage 4 & 5).

Clinical Manifestations are related to the many functions of the kidney:

1. Uremia
2. HyperKalemia
3. Metabolic Acidosis (kidney unable to produce enough ammonia to excrete endogenous acid in the form of ammonium; accumulation of phosphates, sulfates, and other organic acids)

4. Hypoalbuminemia (Acidosis associated with Protein Degradation, Negative Nitrogen Balance, and Decreased Albumin synthesis all of which lead to Protein-Calorie Malnutrition)

5. Malnutrition

6. Renal Osteodystrophy (related to Metabolic Acidosis since bone acts as an acid buffer; decrease in Vit D metabolism to Calcitriol; Secondary HyperParathyroidism)

7. Secondary Hyperparathyroidism (Hyperphosphatemia, Hypocalcemia, Decreased Calcitriol (1,25-Vit D), & Skeletal resistance to PTH) causing very high PTH levels -- contrast this with Primary HPT which has elevated PTH levels but a different electrolyte profile. Secondary HPT usually associated with Hyperplasia of the Parathyroid Gland, not with Adenomas which is typical of Primary HPT.

8. Pulmonary Edema. Volume expansion may cause HTN as well.

9. Anemia due to decreased renal synthesis of Erythropoietin.

10. Bleeding Diathesis secondary to Uremia-induced Platelet Dysfunction.

Now, ACUTE RENAL FAILURE which is what we deal with most of the time in the Critical Care setting. The cause is usually Multifactorial (hypovolemia, hypoperfusion, sepsis, shock, drug toxicity, contrast-toxicity, ATN, obstruction, abdominal compartment syndrome, renal vein thrombosis, etc.). ARF manifests as oliguria (<0.3cc/kg/hr for >/- 24 hrs) and/or rising BUN & Cr.

50% mortality rate associated with ARF in the ICU setting. 15% of those that survive will require dialysis chronically; 85% recover adequate renal function (Dr. Guy rounds).

The goal is to have a system for assessing acute renal failure and/or oliguria when it occurs. Classically, many of us assess based upon a PreRenal, Renal, and PostRenal differential diagnosis or pathophysiology of the process. A classic mistake that occurs in using this approach is to assume that a PreRenal state is a hypovolemic state which requires treatment with more volume. In the SICU, TICU, and BICU this is overwhelmingly the usual situation. However, a PreRenal environment is defined by underperfusion of the glomerulus which may also be associated with HYPERvolemia in patients with heart failure, a patient population that is not as common in the surgical ICUs. Hypervolemia associated with ARF may also be seen in Liver Failure and Nephrotic Syndrome.

Another method for assessing the patient with Oliguria or ARF is by defining the pathophysiology as Mechanical Outflow Obstruction or Decreased GFR.

Mechanical Outflow Obstruction:

1. Tubular Obstruction of the Ureter by stones, papillary slough, crystals, or pigment

2. Urethral or Bladder Neck obstruction by the prostate or by a tumor

3. Urinary Catheter Obstruction

Decreased GFR:

1. Absolute Decrease in Intravascular Volume: Hypovolemia, Hemorrhage, Dehydration, etc.

2. Relative Decrease in Intravascular Volume from CHF or from increased capacitance/vasodilation related to Sepsis, Hepatic Failure, Nephrotic Syndrome, and Vasodilatory Drugs.

3. Decreased Renal Perfusion:

    a. Thromboembolism (esp after thrombolytic therapy)

    b. Atherosclerosis

c. Dissection of Renal Artery

d. Inflammation from Vasculitis, Scleroderma

e. Atheroembolism after manipulation of the aorta or renal artery from Arteriography, Angioplasty, or Surgery

f. Drugs: Cyclosporine, Tacrolimus, ACE-Inhibitors

g. ABDOMINAL COMPARTMENT SYNDROME (Outflow Obstruction of Renal Vein, Direct Renal Compression, Impaired Cardiac Output, Increased Airway Pressures--all of which are detrimental to Renal Perfusion. Ultimately the PERFUSION PRESSURE across the Glomerulus is decreased and Oliguria is the consequence.

h. Renal Vein Thrombosis (outflow obstruction causing an increase in Renal Vein Pressure and therefore a decrease in Perfusion Pressure of the Glomerulus)

4. Acute Tubular Necrosis (ATN) which may be induced by any of the causes above in addition to Nephrotoxic Drugs such as Antibiotics (colistin, aminoglycosides, amphoterocin, vancomycin, etc.), IV-Contrast agents, Heavy Metals, Solvents, Crystals (uric acid & oxalate), Myoglobinuria

After reviewing so many etiologies for ARF, it is easy to understand why ARF is usually MULTIFACTORIAL in the Critical Care setting. Also remember that any of the etiologies may induce ATN which we classically think of as a 'Renal' cause of ARF or oliguria.

Diagnostic & Therapeutic Approach includes:

a. Flush or Change catheter to r/o catheter obstruction.

b. Renal U/S helps r/o upper urinary tract obstruction and vascular etiologies. In upper urinary tract obstruction, Hydronephrosis would likely be identified unless the patient had an absolute or relative superimposed Hypovolemia. U/S also avoids the use of contrast dye in IVP or CT studies.

c. U/A: RBC casts may indicate glomerular disease. Is Myoglobinuria present? Is there a compartment syndrome?

d. Biochemical Indices (unreliable when diuretics or natriuretics are used) such as Urine Osmolality, Urine Na concentration, FENa, FEUrea, Lactate, in addition to others.

e. Evaluate and ensure adequate Volume status. Must maintain adequate Perfusion Pressure of the Glomerulus. In addition to clinical parameters of physical exam, biochemical indices, CVP, Wedge, EDVI, CI, SVO2, CXR findings, other techniques such as IVC diameter and ECHO are also possible although less available and less standardized in the clinical setting.

f. Treat underlying Etiology. For instance, if sepsis is suspected than culture and start antibiotics immediately.

g. D/C nephrotoxic drugs

h. Monitor drug levels

i. Renal dose drugs according to GFR

j. Treat HYPOTENSION (must maintain Perfusion Pressure of the kidney)

k. A higher MAP may be needed in patients with chronic HTN. Their renal autoregulation to maintain perfusion across a wide range of pressures is altered.

l. Decompressive Laparotomy for Abdominal Compartment Syndrome. Laparostomy maintained until resolution of edema permitting abdominal closure.

m. Use diuretics to treat HYPERvolemia. The use of diuretics simply to maintain flow has not been shown to improve outcomes.

n. Low-dose Dopamine has not been shown to improve outcomes--same with other D1 stimulators such as Fenoldopam

o. Consider renal protection with HCO3 or acetylcysteine prior to obtaining contrast studies

p. RENAL REPLACEMENT THERAPY (RRT)--Listen to Dr. Guy's Podcast

- Mechanisms of RRT include Osmosis/Diffusion across a membrane to remove Solute, Ultrafiltration (the movement of Water), Convection (movement of solutes with the movement of water), Hemofiltration (removal of solutes using convection via Ultrafiltration)

- IHD = Intermittent Hemodialysis: generally requires a more stable patient; quick (3-4 hrs); high flows therefore less need for anticoagulation; 20-30% experience Hypotensive episodes during IHD which could aggravate underlying ARF

- CRRT: slower but better HD stability with fewer episodes of Hypotension and indicated in patients with concomitant CNS injury or pathology; disadvantages include the need for anticoagulation (heparin or citrate), clotting of the dialysis filter, and the increased acuity of nursing care

- Forms of CRRT include SCUF (just removes volume, not solutes), CAVHD, CVVHD, CVVHDF, CVVHF, SLED (Sustained Low Efficiency Dialysis) or EDD (Extended Daily Dialysis). SLED/EDD is a hybrid form combining IHD & CRRT.

- PD = Peritoneal Dialysis; not practical for ARF in the Critical Care Setting

Finally, INDICATIONS for DIALYSIS or RRT:

a. Hypervolemia (unable to oxygenate)
b. Uremia
c. Electrolyte problems (HyperKalemia, etc)
d. Acidosis
e. Toxins
f. Internal Warming of Severe Hypothermia

Have a good weekend,

CC

(Sources: Medscape, Critical Care Textbook, Guy Rounds/Podcast)