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RENAL and UROLOGIC EMERGENCIES

Edited by

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and

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Preface

Emergency and primary care physicians encounter disorders of the kidneys and urogenital tract that range from uncomplicated infections to life-threatening trauma and metabolic disturbances. For many of the more serious problems consultation with the nephrologist or urologist will be necessary, but initial evaluation and the execution of any necessary emergency interventions must be the responsibility of the physician who sees the patient first. This volume provides clinicians with the background and information essential for an organized approach to the detection, evaluation, and successful management of renal and urologic disorders in the emergency and primary care setting. It is intended to fill the widely perceived need for a guide and reference which is up-to-date, detailed, and practical, and which provides guidelines for bedside decision-making as well as in-depth expositions of important clinical issues.

The authors are physicians practicing in academic centers who combine a broad range of front-line clinical experience with active scholarly and research interests in their fields. Since the discussions of necessity cannot be encyclopedic and since they have been limited for the most part to the management of adult patients, extensive bibliographies are provided so that the reader may have access to the major sources in the current literature.

We would like to thank the contributors for the considerable effort and time that went into their chapters, and for the high standards which they have brought to their work. For superhuman secretarial assistance, we must express our gratitude to Patricia Laird, Shari Williams, Theresa Haskins, and Julia Meeks; and for consistent and personalized editorial support, to Kim Loretucci of Churchill Livingstone.

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Contents

1 Evaluation of the Azotemic and/or Oliguric Patient

Eugene C. Grochowski

A 70-year-old white female with a history of hypertension and mild compensated congestive heart failure (CHF) enters the emergency department (ED) with a 2-day history of progressive dyspnea. On physical exam her blood pressure is 180/110. She has rales at both lung bases, a soft third heart sound (S_3), distended neck veins, and 2+ pretibial edema. On further questioning she reveals that she "ran out of her water pill"—furosemide (Lasix), 40-mg tabs—4 days ago. Her other medications include digoxin 0.125 mg QD, potassium chloride (Micro K) 8 mEq BID, and ibuprofen (Motrin) 200 mg TID. The physician orders CBC, BUN, creatinine, and electrolytes. She develops a good diuresis—350 ml over the next 2 hours—after an 80-mg dose of furosemide. The laboratory studies reveal: WBC 12,500 per mm^3, hemoglobin 8.5 g/dl, sodium 130 mEq/L, potassium 5.2 mEq/L, bicarbonate 20 mEq/L, chloride 100 mEq/L, BUN 100 mg/dl, and creatinine of 5.0 mg/dl.

What is the etiology of this patient's azotemia? Is this acute or chronic renal failure? Can this patient be safely dismissed from the ED? How soon should she be followed up? What other tests should be done to evaluate this patient's azotemia? Should the patient continue her current medications?

In order to answer these questions, the ED physician must have a systematic approach to the evaluation of the azotemic and/or oliguric patient. The goal of this chapter is to provide such an approach.

First, it will be useful to define the terms azotemia and oliguria:

Oliguria generally is defined as a urine volume of less than 500 ml per day;

assuming a maximum concentrating ability of 1,200 mOsm/L, that is the smallest volume in which the normal daily solute load of approximately 600 mOsm can be excreted.

$$\frac{600 \text{ mOsm}}{0.5 \text{ L}} = 1{,}200 \text{ mOsm/L}$$

However, the maximum concentrating ability is significantly less in the elderly and in patients with chronic renal insufficiency. If the maximum concentrating ability has been reduced to 800 mOsm/L, it will take 750 ml of urine to excrete 600 mOsm of solute.

$$\frac{600 \text{ mOsm}}{800 \text{ mOsm/L}} = 0.75 \text{ L}$$

If the excreted volume is less than these minimum volumes, solutes will be retained.

Azotemia is defined as an increased concentration of nonprotein nitrogen in the blood. This is generally measured as the blood urea nitrogen (BUN). Renal function is usually, but not always, impaired when the BUN is elevated. Conversely, renal function can be impaired without an increase in BUN. Serum creatinine is a better measure of kidney function; the BUN is a better measure of the retained "poisons" when renal failure is known to exist.

The successful approach to the azotemic and/or oliguric patient involves recognizing three distinct etiologic categories of acute renal failure (ARF): prerenal ARF, postrenal ARF, and intrinsic ARF.[1–3] The physician systematically reviews the patient's history, performs a physical examination, and orders laboratory and radiologic studies appropriate to these three broad categories. This approach helps to organize complex clinical problems, and is easy to remember. It will serve as the basic outline in the rest of this chapter.

PRERENAL ARF

This category includes all of the possible causes of decreased renal perfusion. These can be conveniently subdivided into three groups. First is the group of primary cardiac disorders which cause a decrease in cardiac output, e.g., acute myocardial infarction, arrhythmias, myocardiopathies, and valvular disease. The second group includes all the causes of volume depletion, e.g., blood loss, gastrointestinal fluid losses, and renal losses. The third is the diverse group of disorders that cause a redistribution of extracellular fluid (third-spacing): these include the various hypo-albuminemic states, peritonitis, pancreatitis, and loss of peripheral vascular tone.

POSTRENAL ARF

This category includes all of the possible causes of obstruction to urine flow. The obstruction may be at the level of the renal pelvis, e.g., due to stones or sloughed papillae. It can occur at the level of the ureter due to retroperitoneal fibrosis, lymphoma, cancer of the cervix or prostate, or stones. Finally, at the level of the urethra, obstruction may be due to strictures or prostatic enlargement. It is important to remember that obstruction may be severe enough to cause renal failure without causing a noticeable decrease in urine output. On the other hand, the obstruction must involve both kidneys, unless there is only one functioning kidney, in order to cause azotemia.

INTRINSIC ARF

This category encompasses all the renal parenchymal diseases including glomerulonephritis, vasculitis, interstitial nephritis, and acute tubular necrosis (ATN). Although attempts have been made to eliminate this imprecise term,[4] ATN has been used for many years to describe ARF of diverse etiologies that affects the renal tubules. It is a clinical syndrome describing ARF that is neither prerenal nor postrenal ARF, nor any other intrinsic renal disease. Thus ATN is a diagnosis of exclusion.[5] It is characteristically a reversible disorder with variable urine output and with an inability to reabsorb sodium appropriately. The pathophysiology of ATN has been a topic of much animal research and is far from being fully elucidated. Space does not permit a review of this data, but there are several excellent reviews available.[6,7]

HISTORY

Prerenal ARF

A careful patient history serves as a guide for the physical examination and for the judicious use of the laboratory and radiology departments. The major causes of prerenal ARF must be considered as the physician questions the patient.

Intrinsic Myocardial Pump Failure. Does the patient have a history of CHF? What is his current cardiovascular functional ability? How far can he walk without becoming dyspneic? Can be climb a flight of stairs or vacuum a room? Does he have symptoms of angina, paroxysmal nocturnal dyspnea, or orthopnea? Usually, patients with CHF are symptomatic before prerenal ARF supervenes. However, patients with preexisting renal disease may develop marked azotemia with only a mild decrease in cardiac output and thus may have only minimal signs of heart failure.[1]

Volume Depletion. This is undoubtedly the most common cause of azo-

temia and/or oliguria. When there is massive overt blood loss, the diagnosis is obvious. The patient who presents with a massive gastrointestinal (GI) bleed or with major trauma obviously needs blood and fluid replacement in order to improve urine output. Sometimes the blood loss may not be so obvious, however; a ruptured spleen, retroperitoneal bleed, or fracture of a hip or pelvis can cause significant volume depletion that may be overlooked.

Volume depletion can also result from GI losses. Has the patient had recent vomiting or diarrhea? Patients with a history of chronic inflammatory bowel disease should be questioned carefully. They may be so accustomed to frequent watery stools that they could overlook a recent increase in their diarrhea. Sometimes these patients develop mild nausea and simply cannot keep up with their usual GI fluid losses.

Burns are a dramatic cause of fluid loss. Most emergency physicians are familiar with the fluid requirements of burn patients, but it is easy for fluid administration to lag behind requirements, especially in the first few hours following the burn, because of prehospital treatment delays or initial underestimation of the extent of burn damage.

Fluid can also be lost through the kidneys because of diuretic therapy, post-obstructive diuresis, or, very commonly, osmotic diuresis such as that seen in diabetics with hyperglycemia. Some patients with prior renal disease show an inability to concentrate the urine (nephrogenic diabetes insipidus) or have a salt-losing nephropathy. These patients do well until they are unable to take in sufficient quantities of water or salt, for example, because of nausea and vomiting or unconsciousness. The same is true of patients with the central form of diabetes insipidus.

Systemic Vascular Resistance. A significant fall in systemic vascular resistance (SVR) may result in decreased blood pressure (BP) and impaired organ perfusion (prerenal azotemia), despite a normal or increased cardiac cutput (CO).

A decrease in SVR is normally compensated for by an increase in CO in an attempt to preserve BP, but there are limits beyond which CO cannot increase; the result is hypotension. This is the mechanism of the high output failure seen in septic shock. A similar problem may occur during vasodilator therapy for heart failure.

Sepsis should be treated with appropriate antibiotics. Preload should be optimized by judicious fluid administration. Dopamine has been used to increase cardiac output by its $beta_1$-stimulating effects on the heart. When all other treatment modalities fail, alpha-stimulation of the peripheral vasculature with norepinephrine (Levophed) will sometimes raise SVR, increase BP, and improve organ perfusion. Pharmacologic doses of steroids, while not approved by the Food and Drug Administration for use in septic shock, are often used and recommended.[8]

Excessive venodilatation following therapy with nitrates, morphine, nitroprusside, or furosemide may result in a fall in CO because of central venous pooling of blood and decreased venous return to the heart. This is usually easily

correctable with fluid administration. The expanded venous system simply needs to be filled.

A unique form of prerenal azotemia has been reported in connection with the use of the angiotensin-converting enzyme inhibitor captopril (Capoten)[9] in patients with bilateral renal artery stenosis (RAS). In RAS there is decreased flow through the afferent arterioles of the glomerulus. Captopril causes excessive vasodilatation of the efferent arterioles; this reduces glomerular capillary pressure to such a degree that filtration cannot take place. Without filtration there is essentially no kidney function.

Another form of drug-induced prerenal azotemia has been reported with the use of nonsteroidal anti-inflammatory drugs (NSAIDs), agents which inhibit prostaglandin synthesis. Some of the prostaglandins, such as PGE_2 and PGI_2, are vasodilators and modulate the effects of vasoconstrictors such as angiotensin II, norepinephrine, and vasopressin. Although this action seems to be of little importance in healthy euvolemic patients, there are two situations in which prostaglandins appear to be essential for maintenance of adequate renal perfusion.[10–13]

The first situation is the high-renin sodium-retaining state seen in such conditions as congestive heart failure, volume depletion, salt restriction, cirrhosis with ascites, and nephrotic syndrome.[10,11]

The second situation is that of renal insufficiency secondary to intrinsic renal disease, in which renal perfusion may be dependent on the action of intrarenal vasodilator prostaglandins. Use of NSAIDs in this setting has been associated with a reversible decrease in renal function.[11] Avoidance of these agents is not always possible, however. For example, a patient with renal insufficiency secondary to lupus nephritis may require NSAIDs for control of joint symptoms.

Third-spacing. This is the most subtle cause of prerenal ARF. Fluid must be within the vascular tree to perfuse the kidney. Total body salt and water overload, manifested by the presence of peripheral edema, or ascites, does not guarantee that intravascular volume is adequately expanded. In fact, patients with the nephrotic syndrome and edema, or cirrhotics with ascites, often are depleted intravascularly. This depletion can be seriously aggravated by the use of diuretics, which may mobilize fluid out of the vascular tree faster than it can be replaced from the edema or ascitic fluid. In a severely cirrhotic patient, diuretics may precipitate the hepato-renal syndrome.[14]

Patients who have had major surgery or multiple organ system trauma often develop a syndrome of leaky capillaries. When the alveolar capillaries of the lung are involved, the result is adult respiratory distress syndrome (ARDS). When it involves other capillary systems it may cause massive edema and volume depletion.[15]

Large volumes of fluid may accumulate within the lumen of the bowel because of ileus or obstruction. The volume of sequestered fluid is usually not sufficient to cause prerenal ARF, but if the patient is also unable to take

oral liquids or has concomitant vomiting, the combination may result in significant volume depletion.

Intrinsic ARF

There are three facets to the historical evaluation for possible intrinsic renal failure. First, one searches for clues that point to ATN. This is often useful even when a diagnosis of ATN has not been established. For example, a prior history of radiocontrast injection in a diabetic with diabetic nephropathy makes ATN by far the most likely cause of this patient's azotemia and oliguria.[16] An acutely ill hospitalized patient may have a multitude of possible ATN etiologies, but the patient who presents to the ED will ordinarily be less complicated. ATN will be discussed in more detail later in this section.

Second, one should question the patient about symptoms of vasculitis and collagen-vascular disease. Does the patient have a history of arthritis, skin rash, alopecia, hypertension, sinusitis, or edema?[17]

Third, the physician should investigate possible etiologies of chronic renal disease. When a patient with chronic renal disease first presents, it is not always clear whether he or she has acute or chronic renal disease. Hence, it is useful to inquire about some common, easily identifiable chronic renal disorders:

1. Diabetes mellitus: Is the patient a diabetic with a possible underlying diabetic nephropathy? This is a chronic renal disease that may not have been recognized previously. Diabetics also have an increased risk for papillary necrosis, which can cause ARF, and are susceptible to radiocontrast-induced ATN (see below).
2. Polycystic kidney disease: Does the patient have a family history of renal disease? Do any family members have polycystic kidney disease (PCKD)? A family history of death due to kidney failure after the fifth decade, of cerebral vascular accident (CVA) in the third or fourth decades, of intermittent gross hematuria, or of hypertension should suggest PCKD.
3. Alport's syndrome: A family history of deafness may be a clue to this syndrome of hereditary nephritis and deafness that usually results in renal failure by the third or fourth decade in affected males. Females usually do not develop progressive renal failure, but both males and females invariably have a history of microscopic hematuria and may also have a history of intermittent gross hematuria.
4. Analgesic nephropathy: A history of chronic headaches or other chronic pain syndrome may be an important clue to analgesic nephropathy. This is a form of chronic interstitial nephritis caused by excessive use of phenacetin, formerly a very common ingredient in combination analgesic agents, and still available in several popular products. If asked directly, these patients often will deny excessive use of analgesics. A typical patient will have taken four to eight tablets a day for a period of 5 to 10 years, resulting in a cumulative

dose of 2 to 4 kg. Progression of renal disease may be arrested with cessation of analgesic use.

5. Heroin-associated nephropathy: The classic picture is a history of prolonged heroin addiction in association with the nephrotic syndrome. There may be progression to end-stage renal disease over months to years.

ATN

There are a great many causes of ATN; a few of the more common etiologies will be reviewed. It should first be emphasized that when the prerenal state, whatever its cause, is severe and/or prolonged, ATN may ensue. Furthermore, inappropriate treatment may exacerbate the injury. For example, vigorous use of loop diuretics in an attempt to mobilize edema in nephrotic syndrome may further deplete an already depleted vascular volume and so impair renal perfusion that acute tubular necrosis results. Similarly, prolongation of NSAID-induced ARF may lead to local renal ischemia and ATN.[11]

Aminoglycoside toxicity is a common cause of drug-induced ATN.[5,18–20] Azotemia may appear even 1 or 2 weeks following cessation of therapy. Thus a patient who has recently been discharged from the hospital may present to the ED with uremic symptoms secondary to aminoglycoside-induced ATN. The course is highly variable, but usually eventuates in complete recovery in weeks to months. During the recovery phase, these patients may waste potassium and magnesium in the urine because of tubular damage. The nephrotoxic potential of aminoglycosides is high and careful monitoring is essential. Elevated trough levels are better correlated with toxicity than elevated peak levels. Advanced age, prior renal disease, recent previous exposure to aminoglycosides, concomitant use of cephalosporins, and hypokalemia have all ben associated with an increased risk of aminoglycoside-induced ATN,[18] but these risk factors have recently been challenged.[21]

Any acute event that results in hypotension may lead to ATN. Thus, a history of recent surgery, trauma, or cardiac arrest should be sought. These events are found to be the cause of ATN in a hospitalized patient much more frequently than in an ED patient, but such patients may be transferred from one institution to another and may require ED evaluation in the receiving institution. Also, since an increasing number of operations are being performed on outpatients, the possibility of a patient with post-surgical ATN walking into the ED is not unthinkable.

Myoglobin released from damaged muscles and filtered at the glomerulus may cause ATN; the mechanism is unknown. Rhabdomyolysis occurs much more frequently that is recognized;[22,23] myoglobinuria has been seen following crush injuries, acute peripheral vascular occlusion, seizures, hypokalemia, heat stroke, alcoholic stupor, and prolonged coma. If it is recognized at an early stage, ATN can be prevented by prompt intravenous administration of mannitol and bicarbonate.

ATN may be caused in an analogous fashion by intravascular hemolysis