Clinical Management of Renal Failure

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To

BARBARA

Preface

The space allotted to various aspects of renal failure calls for comment. Chronic renal failure accounts for far more morbidity and mortality than does acute. Nevertheless the latter, so often a completely reversible lesion, has been the subject of extensive study during the past decade and a half, and hence has received greater attention in this monograph. Although the methods serving to remove materials from the body are required in only a minority of cases, the fact that no critical evaluation of them has been published explains the considerable detail devoted to this aspect of treatment.

Since this book is intended to give the practicing physician and student a basic foundation for understanding the pathologic physiology of renal failure, its consequences and its treatment, and not to serve as a reference source, no endeavor has been made to append a comprehensive bibliography.

Introduction

Renal failure may be defined as the inability of the kidney, because of intrinsic or extrinsic factors, to carry out its two main functions: 1) the maintenance of a relatively constant composition and volume of the internal environment of the organism and 2) the removal of certain end products of catabolism which are no longer required by the body. Whatever hormonal or blood pressure regulating functions the kidney may have will not be discussed in this monograph.

In the presence of disordered renal function the attempt to maintain a constant "milieu interieur" devolves upon the physician. This he must largely accomplish by care in regulating intake since the kidney is no longer effective in controlling output.

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"Where there are no intelligible indications, it is clear there should be no action."——**John Y. Bassett, M.D., 1850.**

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Acute Renal Failure

ACUTE RENAL SHUTDOWN manifest as oliguria or anuria may result from diverse lesions, some readily reversible, others ultimately reversible, still others, fortunately uncommon, irreversible. The difficulty, often impossibility, of ascertaining whether or not recovery can occur makes it incumbent upon the clinician, faced with a patient who is oliguric or anuric, to proceed on the assumption that the lesion is reversible. This is a fundamental tenet, to be held until conclusive proof as to prognosis becomes available.

OBSTRUCTION

Obviously the first step is to determine that no obstruction exists in the lower urinary tract by the simple expedient of catheterizing the bladder. Next, unless there is virtual certainty as to the cause of renal shutdown, investigation of the upper urinary tract is obligatory. Stones can obstruct both ureters simultaneously or successively, as can neoplasms. A certain number of individuals, either as a result of developmental anomaly or disease, have only one functioning kidney. Obstruction to the ureter of that kidney will lead to apparent renal shutdown (82). A case observed by us is illustrative:

An elderly lady, with known diabetes of many years duration, suffered a myocardial infarction accompanied by a prolonged period of hypotension and followed by extreme oliguria. Although tubular necrosis as a result of

renal ischemia or, less likely, acute papillary necrosis seemed probable, cystoscopy and retrograde pyelography were performed. A small, probably pyelonephrotic right kidney was essentially non-functioning. There was a gush of urine when a stone was dislodged from the lower left ureter and recovery was uneventful.

Another type of obstruction that can easily be overlooked is illustrated by the following case:

A thirty-eight year old man under treatment for giant follicle lymphoma for five years developed anorexia, nausea and lassitude together with moderate ankle and periorbital edema and had gained considerable weight. Urine volume appeared adequate but phenolsulphonephthalein excretion was minimal and no dye could be seen following the intravenous injection of diodrast. The nonprotein nitrogen was 100 mg. per 100 ml. Retrograde pyelography revealed bilateral hydronephrosis and hydroureter with obstruction at the pelvic brim. Following irradiation directed at the retroperitoneal tumor mass the patient had brisk diuresis, lost twenty pounds in weight and was relieved of all symptoms.

This case illustrates the fact that ureteral obstruction, like prostatic obstruction, can cause renal insufficiency despite the presence of a reasonably adequate urine volume.

DEHYDRATION

One of the most common causes of moderately severe oliguria encountered is dehydration, with or without salt depletion. An illustrative case follows:

A middle-aged man with known peptic ulcer developed exsanguinating hematemesis. Multiple blood transfusions restored his blood pressure from shock levels but bleeding from the ulcer continued, necessitating emergency sur-

gery. During the sub-total gastric resection additional blood was administered. The bladder contained only a small amount of concentrated urine twelve hours post-operatively. The diagnosis of acute tubular necrosis due to shock with or without the concomitance of an unrecognized hemolytic transfusion reaction during anesthesia was considered likely. If such were the case fluid administration should be carefully guarded (see below). On the other hand the need for water was suggested by the concentrated urine found in the bladder. A catheter was left in the bladder and one liter of five per cent dextrose in water was administered intravenously. There was a prompt increase in urine flow and with further hydration adequate urine output continued throughout convalescence. No evidence of tubular damage developed.

CAUSES OF TRUE ACUTE "RENAL FAILURE"

True acute renal failure may be defined as any kidney disorder of relatively abrupt development which leads to renal insufficiency, whether as is usually the case accompanied by oliguria or anuria, or not. The use of the term to designate tubular necrosis alone seems unwarranted. The designations "pre-renal azotemia" and "extra-renal azotemia" no longer appear to serve a useful purpose. The main causes of acute renal failure are relatively few: bilateral cortical necrosis, bilateral renal infarction, bilateral hemorrhagic papillary necrosis (or the occurrence of any of these in a solitary previously functioning kidney), acute glomerulonephritis, and acute tubular necrosis. The first two conditions are presumably irreversible. Nevertheless until the diagnosis of an inevitably fatal disorder can be established unequivocally by histologic examination patients in whom these irreparable lesions are suspected should be managed in the same manner as patients whose

disorder is considered self-limited and reversible. Although the precise diagnosis may be suspected there is no certain method of diagnosis other than pathologic examination. Injection of diodrast into the abdominal aorta may render the renal arterial system visible on roentgen ray examination and provide diagnostic aid. However, since this procedure can lead to severe arterial spasm, a highly undesirable event in reversible types of acute renal failure, it can hardly be recommended.

BILATERAL RENAL CORTICAL NECROSIS

Bilateral renal cortical necrosis is most often found in association with disorders of pregnancy, particularly premature separation of the placenta occurring with preeclampsia or eclampsia. An instance of this disorder, unusual because of its occurrence in a male, is summarized:

A sixty-six year old white man was admitted because of lower abdominal pain of two days duration. From his family it was ascertained that he had been treated for hypertension for many years and that he had consumed as much as a quart of whiskey daily. For a month prior to admission he was anoretic, nauseated and vomited frequently. The possibility of ingestion of toxic agents could not be excluded. Examination revealed a dehydrated, restless, confused, somewhat obese, elderly appearing man. Hematocrit was 47 per cent. The blood nonprotein nitrogen was 88 mg. per 100 ml. Leucocytes numbered 20,000 per cu. mm. Serum chloride was 90 mEq./l. No urine was voided. After 3 liters of 5 per cent dextrose in water was administered intravenously his hydration appeared normal but only 25 ml. of frankly bloody urine was obtained. Retrograde pyelograms were entirely normal. During the next ten days he was treated in the manner to be described below (p. 26). One to three ounces of bloody urine were obtained

daily. On the tenth day the nonprotein nitrogen was 225 mg. per 100 ml. and the serum potassium 8.0 mEq./l. Electrocardiograms showed progressive signs of potassium intoxication (see below), with the duration of QRS complexes prolonged to 0.16 second. Since this had developed despite the treatment he was receiving it was apparent that some other modality was required. Following extra-corporeal dialysis* the serum potassium fell to 6.6 mEq./l., the electrocardiogram reverted to a more normal tracing, and the patient appeared symptomatically better. Five days later, after the serum potassium had again risen to 8.0 mEq./l. a second dialysis was performed without accompanying subjective improvement. This was followed by needle biopsy of the left kidney, which in addition to evidences of benign nephrosclerosis, showed apparent necrosis of the entire cortex. Further dialysis was not attempted. Autopsy performed after the patient expired quietly on the twenty-third day of anuria revealed bilateral cortical necrosis of the kidneys and benign nephrosclerosis. Findings consistent with hypertension of long duration were present but there was no obstruction to any of the major renal arteries although all cortical vessels were thrombosed.

Renal biopsy in this case revealed the irreparable nature of the lesion (at least according to current concepts), and indicated the consequent futility of further dialysis.

BILATERAL RENAL INFARCTION

A second type of renal infarction was presented by the following case:

A woman of seventy-two was admitted to another hos-

* Performed at the Peter Bent Brigham Hospital by Dr. John P. Merrill and his associates.

pital complaining of severe left lumbar and flank pain of thirty-six hours duration. A small amount of bloody urine was voided shortly after the onset of pain. Thereafter she was anuric. The past history revealed an attack of similar pain on the right side accompanied by hematuria a year before. Examination revealed hypertension, atherosclerosis of the peripheral arteries, moderate cardiomegaly and left costovertebral angle tenderness. On the fifth day the house officer, receiving a report from the laboratory that the serum bicarbonate was 15 mM/l., infused 100 mM of sodium bicarbonate intravenously. Tetanic convulsions ensued and terminated fatally some hours later.* Autopsy revealed extensive atheromatous changes in the aorta with an old thrombotic occlusion of the right renal artery and a fresh thrombosis of the left renal artery.

Aneurysm of the aorta may similarly lead to obstruction of the renal arteries. Thrombosis of the renal veins or of the inferior vena cava at the level of the renal veins, particularly when it develops suddenly, can produce hemorrhagic infarction of the kidneys. Gradually developing obstruction is said to produce renal cortical atrophy.

ACUTE GLOMERULONEPHRITIS

Acute glomerulonephritis is rarely responsible for *marked* oliguria or anuria. Precipitated by or the sequela of an antecedent group A hemolytic streptococcus infection of one or another specific type, it can most often be recognized from the history and clinical examination of

* The occurrence of tetany presumably was the result of depression of the serum calcium concentration so frequently encountered concomitantly with the elevation of serum phosphate (see p. 21). The chemical acidosis, increasing the ionization of calcium, probably prevented clinical tetany until it was corrected by the bicarbonate infusion. This case illustrates the danger of treating an isolated finding without due consideration for other chemical abnormalities.