Dialysis and the Treatment of Renal Insufficiency

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Foreword

In the United States more than 60,000 patients are treated annually with some form of chronic dialysis. These patients have many unique problems and needs. Health care professionals caring for these patients must understand the pathophysiology of uremia, the principles and mechanics of dialysis, the adjustments of medications required in dialysis patients, the special dietary needs of dialysis patients, the placement and care of vascular access and peritoneal access, and the selection and preparation of dialysis patients for kidney transplantation.

Those involved in dialysis therapies must be familiar with many medical problems in addition to chronic renal failure. Patients with acute renal failure often require dialysis therapy, and these patients also have unique problems and needs. Dialysis therapy and hemoperfusion can be important in the care of drug intoxication and poisoning. Infants and children present challenging dialysis problems.

There are many methods of providing dialysis therapy. Extracorporeal hemodialysis can be offered in many forms with many different types of equipment. Dialysis therapy may utilize the capillaries of the peritoneum via intracorporeal peritoneal dialysis. Conventional dialysis therapies depend primarily on solute diffusion with some contributions of convection. New approaches to body fluid purification may rely more on convective transport of solutes, such as in hemofiltration. Sorbent systems are increasingly employed, such as in hemoperfusion.

The accumulatng scientific literature in all of these fields is quite vast and is increasing. It is no longer easy to be expert in all aspects of body fluid purification therapies.

In this volume Dr. Van Stone and his associates present an extensive collection of information about renal replacement therapy. Dr. Van

Stone has devoted many years to patient care, teaching, and research in the fields so thoroughly covered in this book, and he is well qualified to coordinate this effort. In these times when multi-authored books are more common, Dr. Van Stone has authored 9 of the 16 chapters presented herein. Dr. Van Stone has always approached dialysis therapies with an appreciation and extensive understanding of the state of the art. He has also pursued research to better understand and to improve this expanding and complicated field of medicine. In this book he and the contributing authors share their experiences, knowledge, and insights in a very readable fashion.

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Preface

Over the past 30 years the care of patients with renal insufficiency has become of increasing importance. This is due in large part to the success of hemodialysis in prolonging the life of these patients. In 1982 in the United States alone there were over 70,000 patients being treated for renal failure at the total annual cost of greater than one billion dollars.

The success of this complicated therapy depends on the expertise of members of many disciplines including physicians, nurses, dietitians, social workers, technicians, and others. This book is an attempt to integrate the various aspects of the treatment of renal insufficiency in one volume. (Although renal transplantation is an important aspect in the care of many patients with renal failure, it is not addressed; patients with a successful transplant no longer have renal insufficiency and the treatment of these patients is very different.) The book is intended not only for the practicing physicians, house officers, and medical students involved in the care of patients with renal insufficiency, but also for nurses, dietitians, social workers, technicians, and other paramedical personnel.

In an attempt to avoid excessive duplication and to maintain a relatively consistent style, the majority of this book was written by myself. In order to completely cover the subject, however, I have freely drawn on the expertise of individuals in my own and related disciplines. The theoretical and clinical aspects of peritoneal dialysis are discussed in two excellent chapters by Michael I. Sorkin, M.D. The very important topic of vascular access is covered by W. Kirt Nichols, M.D., who has many years of experience in this field.

The care of the child with renal failure is in many aspects very different from that of the adult and is nicely summarized in the chapter

by Ted Groshong, M.D. Diet therapy is of the utmost importance in the treatment of renal insufficiency. The diet therapy of patients before the need for replacement therapy, of patients undergoing different types of replacement therapy, and of special types of patients such as the diabetic, are reviewed by E. Ann Murray, R.D.

For any treatment of renal insufficiency to be successful, it is extremely important that the patient be as actively involved in his or her own care as possible. In order for this to occur, the patient must have as thorough an understanding of the disease and its treatment as possible, and education of these patients therefore has become very important. The various aspects of patient education are covered by Ann Campbell. Finally, in an era when fiscal restraints are becoming greater, the efficient management of a dialysis facility is becoming mandatory, and these problems are addressed by Judy C. Webb.

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John C. Van Stone

1

Systemic Manifestations of Chronic Renal Failure

All the organ systems of the body are affected by chronic renal insufficiency. Failure of the kidneys can cause severe problems with the cardiovascular system, gastrointestinal tract, bones, and many other organs. The appropriate management of patients with chronic renal insufficiency requires an understanding of the pathogenesis and treatment of these abnormalities. This chapter will discuss the systemic manifestations of chronic renal failure. It is not meant to be an exhaustive review of all the changes which occur in the body secondary to renal insufficiency, but to be a brief review of the various abnormalities with an emphasis placed on the methods and rationale for treating these abnormalities.

ABNORMALITIES IN CALCIUM, PHOSPHORUS, AND VITAMIN D METABOLISM

Abnormalities in the metabolism of calcium, phosphorus, and vitamin D are important not only because they are one of the earliest changes to occur in progressive renal insufficiency, but also because they are treatable. If appropriate treatment is begun early enough, it is usually successful, but if not, these abnormalities can cause severe morbidity and mortality. If untreated, abnormalities in calcium and phosphorus metabolism will lead to renal osteodystrophy with severe bone pain and pathologic fractures, soft tissue calcification with severe pruritus, and vascular calcification which can lead to ischemia or even

gangrene of the extremities. There are three primary abnormalities effecting calcium metabolism which occur in renal insufficiency. These are phosphorus retention from a decrease in phosphorus excretion, decreased calcium absorption secondary to abnormalities of vitamin D activation, and a resistance to the action of parathyroid hormone.

Phosphorus Retention

Phosphorus retention is the earliest problem occurring in chronic renal failure which requires clinical attention.[1] In order to understand phosphorus mtabolism in chronic renal failure it is necessary to briefly review normal phosphorus metabolism. The normal diet contains between 1500 and 2000 mg of phosphorus of which approximately 1000 mg is absorbed from the gastrointestinal tract. To be in neutral phosphorus balance, the normal adult must excrete 1 g of phosphorus each day in the urine. Plasma phosphorus is freely filtered through the glomerulus and variable amounts are absorbed by the renal tubules. Normally, approximately 7 g of phosphorus are filtered through the glomerulus with 85 percent being reabsorbed by the tubules, leaving 1 g to be excreted in the urine.

The two primary methods by which phosphorus balance is normally maintained in the face of differing phosphorus intakes are (1) changes in the total filtered load of phosphorus, mediated through changes in plasma phosphorus concentration and (2) changes in the tubular reabsorption of phosphorus, mediated primarily by changes in parathyroid hormone concentration. As phosphorus intake increases, plasma phosphorus concentration goes up and increases the total amount of phosphorus filtered through the glomerulus. If tubular reabsorption remains constant this in itself will result in an appropriate increase in phosphorus excretion. Phosphorus excretion is also increased, however, by decreasing the amount of phosphorus reabsorbed by the tubules. The major determinant of tubular phosphorus reabsorption is the circulating concentration of parathyroid hormone. As parathyroid hormone levels increase, phosphorus reabsorption by the tubules decreases thus increasing phosphorus excretion.

The effects of renal insufficiency on phosphorus metabolism are schematically illustrated in Figure 1-1. The initial effect of a decline in renal function is a reduction in the filtered load of phosphorus. Filtered phosphorus is equal to the product of the glomerular filtration rate and the plasma phosphorus concentration, therefore, if the glomerular filtration rate decreases by 50 percent, the filtered load of phosphorus

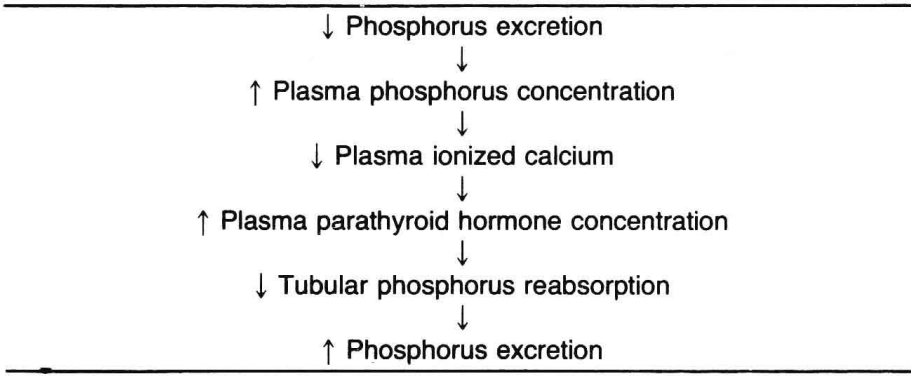

Figure 1-1. Effect of renal insufficiency on phosphorus excretion.

will also decrease by 50 percent. If the tubular reabsorption does not change or even if it decreases in proportion to the decrease in glomerular filtration rate, there will be a reduction in phosphorus excretion with a resultant positive phosphorus balance. The positive phosphorus balance will cause the serum phosphorus to increase. Phosphorus and ionized calcium are in dynamic equilibrium in the plasma and this increase in phosphorus will result in an immediate decrease in ionized calcium concentration. Since the major determinant of parathyroid hormone secretion is the plasma-ionized calcium concentration, there is a rapid increase in parathyroid hormone secretion. The increased plasma parathyroid hormone concentration causes a reduction of tubular phosphorus reabsorption and therefore an increase in phosphorus excretion. This in turn causes plasma phosphorus concentration to decrease and ionized calcium to increase. Early in the course of progressive renal insufficiency this mechanism is sufficient to restore phosphorus balance and maintain both serum phosphorus and serum calcium concentrations in the normal range. These concentrations are only maintained, however, at the expense of a chronic elevation of plasma parathyroid hormone concentration. If untreated, moderate renal insufficiency with a glomerular filtration rate of 20–50 percent of normal, is usually manifested by normal plasma calcium concentration and normal plasma phosphorus concentration, but increased plasma concentrations of parathyroid hormone.[1] These increased parathyroid hormone concentrations have harmful effects on the bones and other organs of patients with renal insufficiency.[2] The chronic stimulation of the parathyroid gland also results in hypertrophy of the gland which makes the later control of secondary hyperparathyroidism more difficult.

Vitamin D Metabolism

Abnormalities of vitamin D metabolism are another major problem in chronic renal insufficiency.[3] Vitamin D is a steroidal compound (Fig. 1-2), normally obtained in the diet. It is also synthesized from cholesterol in the skin after ultraviolet radiation from the sun. The parent vitamin D molecule has very little, if any, direct effects on the body and in order to become activated must have two hydroxyl radicals attached.[4] The first is placed on the 25th carbon position by the liver to form 25 hydroxy vitamin D (Fig. 1-2). The active compound, 1,25 dihydroxy vitamin D ($1,25(OH)_2D_3$), is formed in the kidney by the placement of a second hydroxyl radical on the first carbon position.

$1,25(OH)_2D_3$ can properly be considered a hormone since it is produced by one organ and excreted into the blood stream to have its predominant effects on other organ systems. The rate of production of $1,25(OH)_2D_3$ by the kidney is regulated on the basis of need.[5] The primary function of $1,25(OH)_2D_3$ is to help maintain plasma calcium concentrations in the normal range. The major action of $1,25(OH)_2D_3$ is to increase calcium absorption in the small intestine. $1,25(OH)_2D_3$ stimulates calcium absorption by increasing the synthesis of a calcium carrier protein in the intestinal cells. In the absence of $1,25(OH)_2D_3$ less than 25 percent of dietary calcium may be absorbed, whereas, with high levels of the hormone greater than 75 percent is absorbed.

Vitamin D has other effects in that it plays a permissive role in the action of PTH on bone and may help regulate parathyroid hormone secretion. Vitamin D also affects muscle metabolism, and deficiency can result in severe muscle weakness.[5]

The rate of synthesis of $1,25(OH)_2D_3$ by the kidney is affected in several ways.[6] A low calcium diet, decreased plasma-ionized calcium, increased plasma PTH concentrations, and low serum phosphorus concentration all stimulate $1,25(OH)_2D_3$ synthesis, whereas high plasma calcium and high plasma phosphorus inhibit the synthesis.

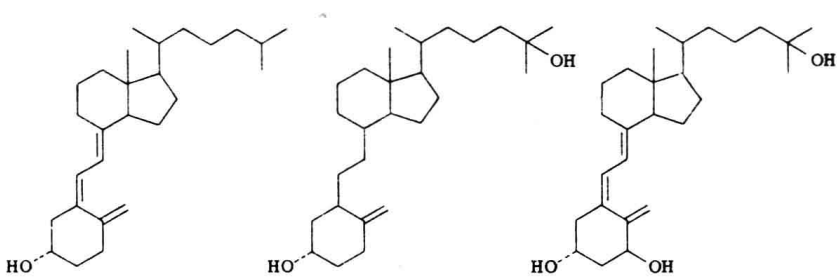

Figure 1-2. Structure of vitamin D and major metabolites.