Experience with Medical Nephrectomy

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INTRODUCTION

In the past, patients with nephrotic syndrome have died from severe proteinuria, hypoproteinemia, anasarca, malnutrition, infection, and end-stage renal disease. The development and effective use of more potent diuretics and antibiotics have improved management of these patients, and treatment by dialysis has prevented death when nephrotic syndrome has gone on to end-stage renal failure and uremia. This prevention of death from renal failure and uremia in these patients has also suggested that, if the degree of proteinuria, edema, and malnutrition were life threatening, the patient’s life might be saved by altering renal function to decrease proteinuria and then maintaining the patient, if necessary, by chronic dialysis. Reports of ablation of renal function for this purpose have described a number of techniques, including surgical nephrectomy and complete infarction of both kidneys by embolization\(^1\)\(^2\) as well as decrease of proteinuria produced either by prostaglandin synthetase inhibitors\(^3\)\(^5\) or by administration of metallic salts\(^6\)\(^7\).

This report details a case in which use of a mercuric salt as described by Avram and co-workers was used successfully to perform a virtually complete ablation of kidney function.

CASE REPORT

C.R., a 26-year-old black man, was first referred to the Ward Teaching Service of the University of Mississippi Hospital on September 18, 1980 by his local physician after failure of steroid treatment (80 mg prednisone daily for about 1 month) to improve his nephrotic syndrome. Prior
to the onset of nephrotic syndrome 2 months previously, the patient had been in good health and worked as a busy cabinet maker. Both of his parents were treated for hypertension, and a grandmother was being treated by hemodialysis in Chicago.

On admission, C.R. was an obese and edematous cushingoid black man of average stature in no acute distress. His blood pressure was 160/90, temperature 98.6°, pulse 80, respirations 18, and weight 90 kg. He showed 4+ pitting edema in the lower extremities and decreased breath sounds with dullness over the bases of both lungs.

Laboratory investigation revealed 4+ protein and many granular casts and oval fat bodies in the urine. His BUN was 31 mg/dl, and serum creatinine was 1.6 mg/dl, serum albumin 1.4 g/dl*, total protein 3.6g/dl, and serum cholesterol 855 mg/dl. Chest X ray was consistent with bilateral pleural effusions. Hematocrit was 37, and white count 11,000 cells/mm³. There was 13 g of proteinuria per day,* and creatinine clearance was 126 ml/min. Studies to demonstrate an etiology of his nephrotic syndrome were negative.

The patient received a diet limited to 87 mEq of sodium per day with maximal intake of protein of high biological value. On September 24, he had a percutaneous renal biopsy guided by excretory urography. The renal tissue obtained showed only fusion of foot processes and was consistent with lipoid nephrosis. Following the biopsy, the patient had severe acute renal failure necessitating hemodialysis via a Scribner–Quinton shunt until October 10. After dialytic therapy and removal of edema fluid, the patient’s weight was reduced to 80 kg. Hospital discharge took place on October 28, 1980, when the serum creatinine was 4.0 mg/dl.

Nephrologists followed C.R. on an outpatient basis until March 18, 1982. He received a diet limited to 87 mEq of sodium, and protein intake was encouraged. Management of edema required increasing doses of furosemide. A rising blood pressure and serum creatinine led to a clinical diagnosis of focal glomerulosclerosis rather than lipoid nephrosis.

He required readmission to the hospital on March 18, 1982 because of pleurisy, fever, and cough productive of yellow phlegm associated with pneumonia and empyema. At this time, his blood pressure was 140/70; he displayed anasarca and weighed 82 kg. His BUN was 81, serum creatinine 8.2 mg/dl, and serum albumin was 0.9 g/dl. Chest X ray suggested an infiltrate in the right lower lung and bilateral pleural effusions. Sputum examination disclosed sheets of gram-negative pleomorphic coccobacilli, some intracellular, suggesting *Haemophilus influen-

*We employed the MacKay modification of the Shevkey–Stafford method to measure proteinuria and the BCG dye binding method to measure serum albumin.