CASE STUDY

A 75-YEAR-OLD MAN WITH DIABETIC NEPHROPATHY

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BACKGROUND
A 75-year-old white man, referred by his primary care physician, presents with a history of type 2 diabetes mellitus (DM), hypertension, slowly progressive chronic renal failure with proteinuria, and a recent elevation in serum potassium (K).

MEDICAL HISTORY
The patient was diagnosed with type 2 DM in 1995 and has had hypertension for more than 20 years. He had transient ischemic attacks in 2002 and 2003. He also suffers from gastroesophageal reflux disease and osteoarthritis of the knees. The patient is currently taking the following daily medications: glipizide, 5 mg; nifedipine, 60 mg; clonidine, 0.1 mg; and clopidogrel bisulfate, 75 mg.

REVIEW OF SYSTEMS
Other than the pain in his knee joints, which is relieved with acetaminophen, the patient has no major medical complaints. He takes no other over-the-counter medications and no nonsteroidal anti-inflammatory drugs. His appetite is excellent; he is particularly fond of fruits, especially bananas, which he eats in large quantities daily. In addition, he drinks fruit juice twice daily, at breakfast and dinner. He has no dysuria and his urinary stream is strong, but he does have increased frequency of micturition and urinates 3 to 4 times at night. Reviews of other systems were negative for disease.

FAMILY HISTORY
The patient's mother had DM and died of heart failure at age 62 years. His father died when the patient was a teenager. One male sibling died at age 69 years from complications caused by high blood pressure (BP).

SOCIAL HISTORY
The patient is a retired truck driver who lives with his wife of 52 years. He has a sedentary lifestyle but enjoys an occasional fishing trip. The patient has not smoked tobacco for the past 20 years. He consumes approximately 6 cans of beer per week.

PHYSICAL EXAMINATION
At presentation, the patient appeared to be alert, well oriented, and obese. Physical examination indicated the following results: weight, 228 lbs; height, 67 inches; BP, 140/85 mm Hg; pulse rate, 71/min. Extremities, 2 plus peripheral edema was present. No skin rashes were apparent. Funduscopic examination revealed no evidence of diabetic retinopathy. His chest was clear to auscultation. His heart rate and rhythm were within the reference ranges, with a soft grade 1 ejection murmur. No jugular venous distention and no carotid bruits were detected. His abdomen was obese, but no abnormalities were evident.

LABORATORY FINDINGS
Findings from the ultrasound examinations of the kidneys and postvoid bladder were within reference ranges. Urine dipstick showed proteinuria (4+). No abnormal sediment was observed in the urinalysis. Spot urine albumin/creatinine ratio was determined to be 3 g albumin/g creatinine. Hemoglobin A1c was 7.0; hemoglobin was 11.5 g/dL. Current blood serum/plasma concentrations were evaluated as follows: sodium, 137 mEq/L (normal); potassium, 5.6 mEq/L (high); chloride, 109 mEq/L (high); carbon dioxide, 22 mEq/L (low); glucose, 55 mg/dL (low); creatinine, 2.7 mg/dL (high); blood urea nitrogen, 55 mg/dL (high); and albumin 3.9 mg/dL (Table).

IMPRESSION AND DIAGNOSIS
The patient is an elderly white man, age 75, with type 2 diabetes mellitus, mild anemia, hypertension, chronic renal failure, hyperkalemia, mild hyper-
chloremic acidosis, and proteinuria. The profile is consistent with diabetic nephropathy. However, because of the absence of retinopathy, it is appropriate to consider other common causes of proteinuric renal disease with bland urinary sediment. In this age group, in a patient with anemia, dysproteinurias should be excluded and a urinary protein immunoelectrophoresis should be requested. Other causes of proteinuric nephropathy with bland urinary sediment to be considered in this age group include focal glomerulosclerosis, membranous nephropathy, minimal change disease, immunoglobulin A nephropathy, and nephrosclerosis. Obstructive uropathy has been excluded by ultrasound examination. In most instances, a renal biopsy would be required to definitely establish the renal diagnosis, but based on the chronic nature and severity of the patient’s renal impairment, age, and general condition, a renal biopsy will not be considered. The hyperkalemia and nonanion gap hyperchloremic acidosis (type IV renal tubular acidosis) may be caused by hyporeninemic hypoaldosteronism, of which diabetic nephropathy is the most common cause.

**TREATMENT PLAN**

Vigorous BP control is required with the target BP of 130/80 mm Hg or even lower if heavy proteinuria persists. Because hyperkalemia is present, blockade of the renin-angiotensin system is contraindicated and a nondihydropyridine calcium channel blocker, such as diltiazem, should be instituted instead. The diuretic dose should be increased to correct the fluid and K⁺ retention and to improve BP control. The dose of clonidine could be increased if optimal BP control is not achieved. The patient was counseled to reduce his intake of fruit and other high-K foods and protein, and a visit to a nutritionist was arranged. Repeat serum K evaluation after 5 days was 5.1 mEq/L.

<table>
<thead>
<tr>
<th>SER/PLA</th>
<th>NA⁺</th>
<th>K⁺</th>
<th>CL</th>
<th>CO₂</th>
<th>GLU</th>
<th>CREAT</th>
<th>BUN</th>
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<tbody>
<tr>
<td>Ref range</td>
<td>136–145 mEq/L</td>
<td>3.5–5.5 mEq/L</td>
<td>96–108 mEq/L</td>
<td>24–32 mEq/L</td>
<td>70–110 mg/dL</td>
<td>0.8–1.4 mg/dL</td>
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<td>5.6 H</td>
<td>109 H</td>
<td>22 L</td>
<td>55 L</td>
<td>2.7</td>
<td>55</td>
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<tr>
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<td>28</td>
<td>137 H</td>
<td>1.9</td>
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</tr>
</tbody>
</table>

BUN = blood urea nitrogen; CL = chloride; CO₂ = carbon dioxide; CREAT = creatinine; GLU = glucose; H = high; K⁺ = potassium; L = low; NA⁺ = sodium; PLA = plasma; SER = serum.