Evidence-Based Clinical Vignettes from the Care Management Institute:

A Diabetic Patient with Renal Disease and Heart Failure

In previous issues of The Permanente Journal, our Clinical Vignettes have each highlighted a single disease state for which the Kaiser Permanente Care Management Institute recently developed evidence-based guidelines after systematic literature review. In this issue, we describe a hypothetical patient with multiple medical comorbid conditions—a situation most of us see every day. We asked several physicians to help outline an evidence-based treatment plan. Our respondents include:

- Jim Dudl, MD, Endocrinology, Southern California Permanente Medical Group, and Care Management Institute Clinical Lead for Diabetes.
- Tony Steimle, MD, Cardiology, The Permanente Medical Group and Care Management Institute Clinical Lead for Heart Failure.
- Mohamed Idroos, MD, Nephrology, Southern California Permanente Medical Group.
- David Sobel, MD, Primary Care Medicine; Director of Patient Education, The Permanente Medical Group; and Physician Co-Lead for Care Management Institute Self-Care and Shared Decision-Making.

The case presenter is Antoine Abcar, MD, Nephrology, Southern California Permanente Medical Group.

A 44-year-old man is seen in the adult primary care department as a new patient. He has a 32-year history of Type I diabetes mellitus and a two-year history of dilated cardiomyopathy (nonischemic). Results of previous evaluation of the cardiomyopathy include a normal coronary angiogram. The most recent echocardiogram, taken four months previously, shows normal valvular anatomy and an ejection fraction of 25%. About six months previously, an optometrist told the patient that he had mild-to-moderate diabetic retinopathy. The patient has had no previous surgery.

The patient has no known drug allergy. He does not smoke, drink alcohol, or use recreational drugs. He denies use of nonsteroidal anti-inflammatory or over-the-counter medications. His current medications include 70/30 insulin (25 U in the morning and 15 U at dinnertime), lisinopril (10 mg/day), and carvedilol (6.25 mg twice daily).

His parents and two of his brothers have hypertension; his father, one brother, and one sister have diabetes mellitus. His father was also diagnosed with coronary artery disease in his early 50s. The patient has no known family history of stroke, cancer, rheumatic disease, nephrolithiasis, hematuria, proteinuria, or renal failure.

He states that he feels well in general. His blood glucose values range between 105 mg/dL (5.8 mmol/L) and 245 mg/dL (13.6 mmol/L). He says he has no chest pain, shortness of breath at rest, or hematuria. Urine output has been good, but he has occasionally noted lower-extremity edema late in the day.

He seems mildly anxious about visiting a doctor but is otherwise in no apparent physical distress. He weighs 205 pounds and is 70 inches tall. Blood pressure measured in the left arm with the patient seated is 145/86 mm Hg. The pulse is 64 beats per minute. Funduscopic examination is difficult to perform. The oral mucosa is moist. Results of thyroid examination are normal, and no cervical bruit is heard. The lungs are clear. No heart murmur is detected except for possible intermittent S3 gallop. The abdomen is soft without organomegaly, mass, or bruit. Results of extremity examination are normal except for trace ankle edema. The patient has no foot ulcers, and the dorsalis pedis pulse is barely palpable bilaterally.

After obtaining the above baseline history and physical examination values, you order diagnostic tests (results are summarized in Table 1) and arrange for the patient to return for follow-up in two weeks. Complete blood count, as well as results of thyroid and liver function tests are normal. A chest x-ray film shows an enlarged cardiac silhouette without pleural effusion. An electrocardiogram shows sinus rhythm with left ventricular hypertrophy and left axis deviation.

Describe your general approach to the patient:

Dr Dudl: I’d start by thinking about the risk of complications and then what we can do to prevent them. As noted in our previous article in The Permanente
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I would let him know that I look forward to establishing a long-term partnership with him and to working with him to better manage these conditions. I would point out some of the ‘good news’ about his health condition (eg, normal coronary angiogram, lungs clear at examination, normal serum creatinine level) while expressing my confidence that some areas could be improved and that we could better manage some problems (eg, HbA1c level, CHF).

Dr Sobel: The patient has a complex medical history, and several visits are needed to understand his problems, life situation, and what he is doing to self-manage his conditions. I would let him know that I look forward to establishing a long-term partnership with him and to working with him to better manage these conditions. I would point out some of the ‘good news’ about his health condition (eg, normal coronary angiogram, lungs clear at examination, normal serum creatinine level) while expressing my confidence that some areas could be improved and that we could better manage some problems (eg, HbA1c level, CHF).

What additional history would you elicit at the patient’s follow-up visit?

Dr Idroos: I’d want to ask how he monitors his disease at home: Does he check his blood pressure at home? His weight? His feet? How often does he check his blood glucose level? I’d ask about symptoms of autonomic or peripheral neuropathy—nausea, vomiting, dizziness, and numbness of the extremities.

Dr Steimle: I’d also ask him about his dietary history, particularly regarding salt intake and also about exercise capacity. Dyspnea with exertion and especially orthopnea are important clues regarding fluid status.

Dr Dudl: Eliciting a history of diabetic ketoacidosis or hypoglycemic episodes is important to tell whether a coma prevention strategy is needed.

Dr Sobel: I would want to develop a better understanding of his priorities and concerns, the effect of his illness on his life, how well he is self-managing his medical conditions, and what motivates him. I might ask such questions as “What concerns or worries you most about your medical conditions?” and “How are your health problems affecting your daily activities, work, and family?” as well as “What are you hoping I can help you with?” I’d also like to know more about his social history, including work and family.

What additional physical examination should you perform?

Dr Dudl: Anyone with nephropathy and retinopathy is at high risk for neuropathy. We are not given the results of monofilament testing of the feet, but this testing should be done. If sensation to monofilament is absent at 10 g of pressure (a 10 g monofilament is vertically applied to specific points on the foot until monofilament begins to buckle) and if the patient has foot deformity or amputation, risk of amputation is nearly 20 times that of diabetic patients who do not have these signs or history.2 The presence of peripheral vascular disease will also clinically significantly elevate the risk of amputation.3 You would need to use caution about overinterpreting the edema because it could be from decreased oncotic pressure due to proteinuria.

Dr Steimle: To help determine whether the edema is from fluid overload, physical examination should assess jugular venous pressure.4

Dr Idroos: In addition to checking for peripheral neuropathy, orthostatic blood pressure and pulse could provide clues to autonomic neuropathy.

What additional diagnostic testing should be considered? Why?

Dr Dudl: I would think through the causes of CHF to determine whether other testing is needed. In this case, CHF was not caused by myocardial infarction or valvular disease. The patient has a family history of hypertension, his systolic blood pressure is still 145 mm Hg while taking two antihypertensive medications, and his electrocardiogram reveals left ventricular hypertrophy—making hypertension a likely contributing factor to the CHF. Whether or not “diabetic cardiomyopathy” is a factor is not clear to me, and I defer to Dr Steimle regarding diagnosis and treatment. However, a recent review found that “diabetic cardiomy-


table 1. Results of clinical diagnostic tests for hypothetical diabetic patient with renal disease and heart failure

<table>
<thead>
<tr>
<th>Test</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>Serum creatinine</td>
<td>1.4 mg/dL (123.8 mmol/L)</td>
</tr>
<tr>
<td>Serum urea nitrogen</td>
<td>30 mg/dL (10.7 mmol/L)</td>
</tr>
<tr>
<td>Sodium</td>
<td>135 mEq/L (135 mmol/L)</td>
</tr>
<tr>
<td>Potassium</td>
<td>4.8 mEq/L (4.8 mmol/L)</td>
</tr>
<tr>
<td>Chloride</td>
<td>102 mEq/L (102 mmol/L)</td>
</tr>
<tr>
<td>Bicarbonate</td>
<td>27 mEq/L (27 mmol/L)</td>
</tr>
<tr>
<td>Serum cholesterol:</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>215 mg/dL (5.56 mmol/L)</td>
</tr>
<tr>
<td>High-density lipoprotein</td>
<td>44 mg/dL (1.14 mmol/L)</td>
</tr>
<tr>
<td>Low-density lipoprotein</td>
<td>152 mg/dL (3.93 mmol/L)</td>
</tr>
<tr>
<td>Triglycerides</td>
<td>94 mg/dL (1.06 mmol/L)</td>
</tr>
<tr>
<td>Glycosylated hemoglobin (HbA1c)</td>
<td>8.9% of total hemoglobin</td>
</tr>
<tr>
<td>Urinalysis:</td>
<td></td>
</tr>
<tr>
<td>Protein</td>
<td>1+ proteinuria</td>
</tr>
<tr>
<td>Microscopic analysis</td>
<td>0-4 red blood cells/high-powered field</td>
</tr>
<tr>
<td></td>
<td>0 white blood cells</td>
</tr>
<tr>
<td></td>
<td>0 cellular casts</td>
</tr>
<tr>
<td>24-hour urine total protein</td>
<td>855 mg</td>
</tr>
</tbody>
</table>

I would let him know that I look forward to establishing a long-term partnership with him ...
opathy” is a condition that poses clinically significant risk to patients and necessitates further treatment.

The patient’s coronary arteriogram was described as normal. I would, however, review the report or films because at times “normal” means no lesion with more than 30% to 75% luminal occlusion evident, and I would expect early lesions in a patient who has renal disease and a 32-year history of diabetes. The decreased pulses suggest presence of peripheral vascular disease; however, the decreased ejection fraction and beta blockade may be contributing factors. For confirmation, I would order noninvasive vascular studies of the lower extremities. However, even if no evidence of atherosclerosis were present, I would conclude (because presence of diabetes and renal disease increases risk for mortality) that he has as high a risk of a having a CVD event in the next ten years as someone who already has had myocardial infarction.

**What medication adjustments would you initiate?**

**Dr Dudl:** Because this patient is at high risk for CVD, I would focus on CVD prevention using the AABBCC'S mentioned in our prior article: Aspirin, Angiotensin-converting enzyme inhibitors (ACE-I), Beta-blockers, Blood pressure control, and glucose control.

**Dr Idroos:** The main objectives of therapy in this patient are to maximize cardiac function, to obtain tight glucose control (HbA1c<7%), and to aggressively lower the low-density lipoprotein cholesterol level to <100 mg/dl by using a statin. I would gradually increase lisinopril to a maximum daily dosage of 40 mg, gradually increase carvedilol to a maximum of 25 mg twice daily, and add spironolactone at 25 mg daily for the antialdosterone effect instead of for the diuretic effect. Furosemide will help prevent hyperkalemia resulting from hyporeninemic hypoaldosteronism caused by diabetes and from use of an ACE-I and spironolactone. I would consider using digoxin if needed for relief of CHF symptoms.

**Dr Steinle:** I would increase the carvedilol to 12.5 mg twice daily on the first visit and eventually maybe even to 25 mg twice daily (titrated slowly when the patient is euolemic). I agree with increasing the lisinopril to 40 mg daily. If assessment of jugular venous pressure suggests fluid overload, I may need to add a diuretic agent, but I would increase the lisinopril first—sometimes it’s easier to increase the ACE-I dosage when patients are just “a touch wet.” However, if the neck veins are really distended and he has acute symptoms of CHF, I would probably add furosemide now. After titrating the lisinopril, if the serum potassium and creatinine levels remain stable, I would add spironolactone at 25 mg daily. Spironolactone could be added earlier for its weak diuretic effect to counteract mild fluid retention not severe enough to require furosemide. If the patient is not taking furosemide but remains hypertensive despite these medications, I would think about adding either hydrochlorothiazide (HCTZ), as recommended by the ALLHAT Collaborative Research Group, or hydralazine.

**Dr Sobel:** Before adjusting any of the medications, I need to better understand this patient’s current adherence. I might say, “Sometimes it is difficult to remember to take your medication. Have you found this a problem? How often in a week do you forget? Have you been having any side effects or problems taking your medication?” I would acknowledge that he has a lot of different medications to manage and that I can work with him to make sure he understands how these medications are intended to help him as well as how to manage his complex medication regimen within his busy life. If I recommend that he change a medication or start a new one, I might ask, “Do you anticipate any problems with this treatment plan? What do you think you could do to help overcome them?”

**Dr Dudl:** I would start lovastatin at 40 mg daily, per our new Southern California Permanente Medical Group guidelines. This patient’s risk of CVD is similar to that of a person who has had myocardial infarction, and his low-density lipoprotein cholesterol (LDL-C) level is above the target level of 100 mg/dL (2.59 mmol/L). I would use caution going above 40 mg of lovastatin, however, because of increased potential for rhabdomyolysis in patients with renal disease. If creatinine clearance rate falls below 30 mL/min (0.50 mL/sec), then 20 mg of lovastatin daily is the maximum recommended dosage.

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with 10 U of Lente zinc suspension insulin at bedtime and would teach the patient to increase the dose by 1 U every other night until his fasting blood glucose level is 80 to 140 mg/dL (4.44-7.77 mmol/L). Similarly, I would start with 10 U of Lente insulin in the morning and would increase the dose by 1 U every other day until the presupper glucose levels are in the same range. I’d also start a regimen of regular insulin of 5 U at breakfast and 5 U at supper and would then have the patient adjust this dose to keep his blood glucose levels to within the same range (80-140 mg/dL; 4.44-7.77 mmol/L) four to six hours after breakfast and dinner. To achieve this control, I would instruct the patient to use a sliding scale that adds one unit of regular insulin for every 40 mg/dL above 100 mg/dL.

What other resources are available to help you care for this patient? What self-care strategies can be used?

**Dr Steimle:** I would ask him to attend our heart failure classes (available at KP Northern California) and to participate in our heart failure management programs. I would also advise the patient to weigh himself daily and to exercise for his poorly controlled diabetes.

**Dr Dudl:** I would have the patient buy a blood pressure cuff and learn to adjust antihypertensive medication doses himself to maintain systolic blood pressure between 110 and 130 mm Hg. Glucose control is correlated with frequency of glucose testing, so I would emphasize that the patient check his glucose level at least four times daily until glucose control is achieved. Education in our insulin adjustment classes or with a diabetic nurse educator is often very helpful. If he is insensate to the monofilament at 10 g of pressure or has clinically significant peripheral vascular disease, he should receive education about foot care and/or have regular foot follow-up, each of which decreases the risk of amputation. Finally, close monitoring by an ophthalmologist is mandatory, because eye disease can worsen during the first two years of improved glucose control.

This information is a lot for a patient to absorb and could be frightening as well as discouraging. What words would you actually say to the patient to give him hope? How would you help him figure out where to start?

**Dr Sobel:** I would begin by acknowledging his efforts and success thus far in self-managing his complex medical conditions. I would assess how much he already knows about his conditions, home monitoring, prognosis, etc, and would praise, whenever appropriate, what he is already doing. I would find out what concerns him the most and what, if anything, he would like to change (diet, exercise, stress management, or nothing). Depending upon his medical need, interest, and motivation, I would offer him choices or resources, which might include the health information resources, discussion groups, and health encyclopedia available online to Kaiser Permanente members at (http://kp.org), patient education classes, and care management programs as appropriate. Above all, I would communicate realistic optimism and the need for a long-term partnership. I might say, “Although you have many complex medical problems, I am confident we can work together to help you feel better and to prevent complications. Many things can be done to improve your health and to lower your risk, and we can do them step by step.”

Dr Dudl: I would tell this patient, “Some patients are frightened by their diabetes. However, every potential complication we have discussed has something you can do to prevent it. I am confident that if you do your share to improve glucose control and take the combination of medications as advised, you will decrease the risk of those complications and will feel good about yourself by taking charge.”

**References**

4. Steimle A. Evidence-based clinical vignettes from the Care Management Institute: A Diabetic Patient with Renal Disease and Heart Failure.


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The Doctor Inside

Each patient carries his own doctor inside him. They come to us not knowing that truth. We are at our best when we give the doctor who resides inside each patient the chance to go to work.

Albert Schweitzer, 1875-1965, philosopher, physician, musician, 1951 Nobel Peace Prize winner