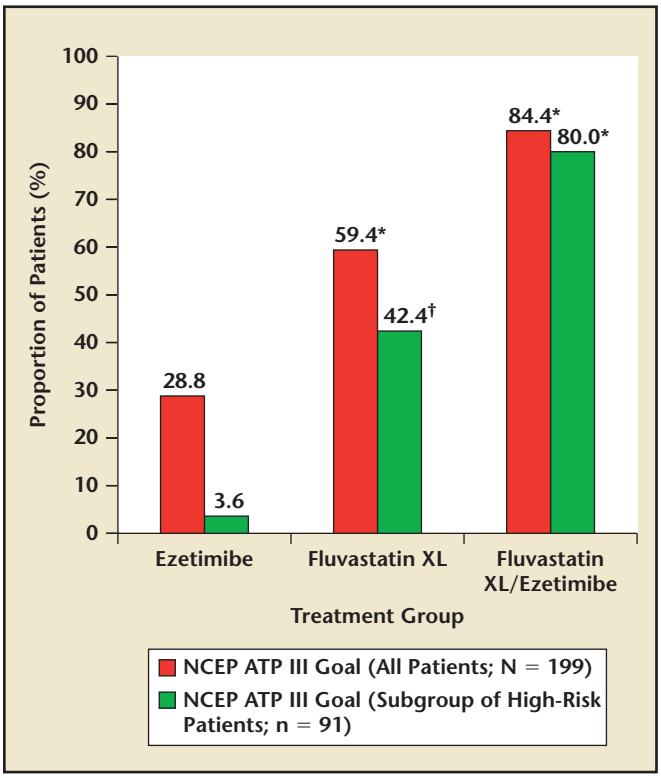

Figure 3. *Proportion of patients achieving their National Cholesterol Education Program Adult Treatment Panel III (NCEP ATP III) low-density lipoprotein cholesterol goals (< 100 mg/dL for high-risk patients, < 130 mg/dL for moderate-risk patients, and < 160 mg/dL for low-risk patients) at week 12. *P < .001; †P = .002 vs ezetimibe monotherapy. Reprinted from* Journal of the American College of Cardiology. *Volume 101, Stein EA et al. Managing dyslipidemia in chronic kidney disease. Pages 490-496.*[8] *Copyright © 2008, with permission from the American College of Cardiology.*

Diabetes Mellitus

Detection of Ischemia

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Cardiac Outcomes After Screening for Asymptomatic Coronary Artery Disease in Patients With Type 2 Diabetes: The DIAD Study: A Randomized Controlled Trial

Young LH, Wackers FJ, Chyun DA, et al.

JAMA. 2009;301:1547-1555.

The Detection of Ischemia in Asymptomatic Patients With Diabetes (DIAD) study was a randomized trial of 1123 patients with type 2 diabetes mellitus (T2DM) with no symptoms of or previously recognized coronary artery disease, who were assigned to undergo screening with adenosine-stress myocardial perfusion imaging or to not undergo screening.[9] The primary endpoint of the study was the incidence of cardiac death or nonfatal MI. The mean follow-up period was 4.8 years. Patients were excluded if they had angina, a stress evaluation or coronary angiogram within the last 3 years, abnormal resting electrocardiogram, or any clinical indication for a stress test. The mean age of the patients enrolled in the study was 61 years, and the average duration of diabetes was just over 8 years. This was a low-risk population of T2DM; only 6% of patients had large or medium-sized perfusion defects, 10% had small perfusion defects, and 6% had nonperfusion defects (eg, abnormal stress electrocardiogram, transient ischemic dilation). A cardiac event was reported in 12% of patients with moderate to large perfusion defects, in only 2% of patients with normal or small defects, and in 6.7% of patients with nonperfusion defects. During the follow-up period, 30% of patients underwent a nonprotocol stress test due to a clinical indication.

The results of this evaluation are not surprising in the least. The only way a screening examination will lead to a reduction of cardiac risk is if it leads to initiation of life-saving therapy. In this clinical trial, there was no difference in the use of lipid-lowering drugs, statins, angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, or aspirin either at the initiation of the clinical trial or at completion. Abnormal stress tests may have led to revascularization procedures, which, in this low-risk, asymptomatic, stable T2DM patient population, have not been shown to be life-saving.

Is a screening nuclear perfusion examination the correct test to perform in this low-risk patient cohort? Instead, should we be assessing coronary risk by using coronary calcium screening, carotid intimal media thickening (cIMT), or carotid artery assessments with magnetic resonance imaging, which do a much better job of identifying those patients who have subclinical coronary artery disease? Or should we not be screening at all? Only if screening were to impact the selection or intensity of life-saving therapies should it be performed because it is not the screening examination that saves lives but the actions that result from it. A good example would be the 40-year-old woman with T2DM who has an LDL-C of 125 mg/dL, who is not on any prevention therapies, and who then has an abnormal cIMT. Or the 45-year-old man

who was recently diagnosed with T2DM, who had a normal cIMT followed by a coronary calcium score that was abnormal, and who was told by his primary care physician that his LDL-C of 130 mg/dL and high-density lipoprotein cholesterol (HDL-C) of 35 mg/dL were "ok." Should we screen the patient with T2DM who is already taking 81 mg of aspirin and a statin and has an LDL-C of 65 mg/dL? Probably not, because an abnormal score would not lead to an intensification of therapy.

A powerful argument can be made that screening modalities can enhance our ability to risk stratify patients beyond the Framingham Risk Score. They can also be a powerful tool to motivate patients and the treating physician. Because a stress test only screens for obstructive coronary artery disease, it would not seem to be an appropriate approach in the presence of any coronary artery disease. ■

Dyslipidemia

Treatment in Patients With Chronic Kidney Disease

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Table 1
Dosing Modifications for Lipid-Lowering Drugs in CKD

Agent	GFR 60-90 mL/min/1.73 m^2	GFR 15-59 mL/min/1.73 m^2	GFR < 15 mL/min/1.73 m^2	Notes
Statins				
Atorvastatin	No	No	No	
Fluvastatin	No	Not defined	Not defined	↓ dose to one-half at GFR < 30 mL/min/1.73 m^2
Lovastatin	No	↓ to 50%	↓ to 50%	↓ dose to one-half at GFR < 30 mL/min/1.73 m^2
Pravastatin	No	No	No	Start at 10 mg/d for GFR < 60 mL/min/1.73 m^2
Rosuvastatin	No	5-10 mg	5-10 mg	Start at 5 mg/d for GFR < 30 mL/min/1.73 m^2, maximum dose 10 mg/d
Simvastatin	No	No	5 mg	Start at 5 mg if GFR < 10 mL/min/1.73 m^2
Nonstatins				
Nicotinic acid	No	No	↓ to 50%	34% kidney absorption
Cholestyramine	No	No	No	Not absorbed
Colesevelam	No	No	No	Not absorbed
Ezetimibe	No	No	No	
Fenofibrate	↓ to 50%	↓ to 25%	Avoid	May ↑ serum creatinine
Gemfibrozil	No	No	No	NLA recommends a dose of 600 mg/d for GFR 15-59 mL/min/1.73 m^2 and avoiding use for GFR < 15 mL/min/1.73 m^2
Omega-3 fatty acids	No	No	No	

CKD, chronic kidney disease; GFR, glomerular filtration rate; NLA, National Lipid Association.
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