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Clinical Decision Points:
Profiles in Patient Care for Nurse Practitioners and Physician Assistants
Based on a Series of National CME/CE Symposia

Q&A: Mixed Dyslipidemia

What Your Colleagues Around the Country Want to Know...

Q: Can very low low-density lipoprotein cholesterol (LDL-C) be harmful?

A: This is a difficult question to answer because some studies suggest that lower LDL-C values are better, but there are no guidelines that specify how low to go. Is <60 mg/dL better than <70 mg/dL? Results of 1 study showed that patients with LDL-C as low as 40 mg/dL did well. If a patient starts trending below that, it's probably reasonable to decrease the dose of the lipid medication being prescribed. However, there is no benchmark study that shows why LDL-C <40 mg/dL is not good. For example, we know there likely is a lower level for diastolic blood pressure that should not be exceeded. We don't have that information for LDL-C.

Q: If a patient had a myocardial infarction (MI) or other cardiac event, is it safe to assume peripheral vascular disease exists as well?

A: Cardiovascular diseases (CVDs) tend to cluster, so when a patient presents with one, it is wise to proactively look for others you suspect, rather than waiting for a CVD to present itself. It might be prudent to obtain an ankle-brachial index, listen carefully for femoral bruits, or take a careful history for claudication. This is an opportunity for preventive care. If a patient has coronary disease, listen to the carotids carefully. Conversely, if a patient has carotid disease, there's always a concern about coronary disease. It's the same case with peripheral vascular disease, because the risk factors are basically the same. If a patient has multiple cardiovascular risk factors, it's essential to perform a thorough evaluation and provide comprehensive therapy to address each one.

Q: When is it appropriate to use prescription omega-3s to treat elevated triglycerides (TGs)?

A: In addition to being an important option in treating hypertriglyceridemia, omega-3 fatty acids have demonstrated other benefits in clinical trials. The Gruppo Italiano per lo Studio della Sopravvivenza nell'Infarto Miocardico (GISSI)¹ trial demonstrated a reduction in sudden death in



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patients following an acute MI who were treated with omega 3 fatty acids. Over-the-counter fish oils are another treatment for hypertriglyceridemia, but a very high dose is required to produce a change in TG values. While prescription omega-3s are more expensive, they are more convenient and lower doses are required to reduce TGs. The Japan EPA Lipid Intervention Study (JELIS)² looked at the effects of eicosapentaenoic acid (EPA) on cardiovascular events in patients with hypercholesterolemia plus multiple risk factors who were on statin therapy, but had no evidence of coronary artery disease (CAD). In the higher risk group (TG ≥ 150 mg/dL, high-density lipoprotein cholesterol [HDL-C] < 40 mg/dL), EPA treatment added to statin therapy suppressed CAD risk 53%.²

Q: What is the association between niacin and gout?

A: Gout is one of the adverse events that may occur with niacin treatment, which is unfortunate because niacin can produce excellent results in appropriate patients with hyperlipidemia. In a patient who had gout or is prone to frequent bouts of gout, the risk/benefit ratio has to be carefully calculated. Allopurinol will address the elevated uric acid, and it may be worth a trial of a combination of the 2 agents, but patients should be selected carefully and closely monitored.

Niacin also affects glucose metabolism, particularly at higher doses. In a patient with controlled diabetes who monitors glucose reliably, niacin would be an appropriate choice. If A1C is out of control and the patient is not self-monitoring at home, niacin may not be the best choice, at least not at higher doses. This issue highlights why it is important for primary care clinicians who treat cardiovascular risk not to become overly focused on a single risk factor—lipids, *or* glucose, *or* hypertension, *or* obesity. There is a lot of “cross-talking” going on with diseases and medications. It’s our job to understand all of the interactions.

Q: If niacin 500 mg brings TGs to goal, is it prudent to increase the dose to 1000 mg or 1500 mg?

A: When TG goal is reached, it generally is not necessary to titrate to a higher dose. But while 500 mg may impact TGs, that dose probably will not achieve the HDL-C goal and a higher dose will be required. In a patient approaching therapeutic goal, the niacin dose can be pushed to 1000 mg,



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or even 1500 mg, if necessary. If side effects occur, the dose should be reduced. The same is true with statins. If the patient is tolerating atorvastatin 40 mg, but is complaining of clinically significant muscle pain at 80 mg, it's time to cut back. There is usually no question theoretically that a dose increase will enhance results, but the clinical picture is more important. If a patient is flushing on niacin or aching on statin therapy, it's likely they will stop taking the medication if those problems aren't addressed through a reduction in dose.

Q: When a patient complains of pain associated with initiation or titration of a statin, what is the appropriate course of action?

A: The best tool at this point is question and answer with the patient. The history is important because many patients have read or heard about the side effects associated with statins and will automatically assume an association between the drug and a symptom. If a patient stops taking a statin because "it made me hurt," find out where they hurt. First it's a good idea to rule out musculoskeletal conditions. If the pain is in the knees and the knees are obviously arthritic, the statin probably isn't the problem. However, if the index of suspicion is still high and if symptoms are severe, it's best to stop therapy and order a creatinine kinase (CK) level. If the CK is elevated, decrease the dose or hold the medication. It's not uncommon, however, to have a normal CK while symptoms persist. The best approach is talking it through with the patient. A plausible scenario might proceed this way: "I take atorvastatin 40 mg. I can't tolerate simvastatin—it makes me ache, and it's real—and I can't take atorvastatin 80 mg. I had to use trial and error to find the dose I can tolerate. I put up with a little bit of muscle pain because I know the positive outweighs the negative. But if atorvastatin caused the same symptoms I had with simvastatin, I'd have to stop it because I would be miserable." For some patients it will become a quality of life issue, and that is legitimate. The clinician-patient relationship is important when treating lipid abnormalities—every patient is different and has a distinct set of circumstances. It may take time to find the right treatment, so ongoing communication is the key.



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References

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2. Saito Y, Yokoyama M, Origasa H, et al; JELIS Investigators. Effects of EPA on coronary artery disease in hypercholesterolemic patients with multiple risk factors: sub-analysis of primary prevention cases from the Japan EPA Lipid Intervention Study (JELIS). *Atherosclerosis*. 2008;200:135-140.