


The Official Publication of the National Lipid Association

LipidSpin

■ **Clinical Feature**

Severe hypertriglyceridemia



Also in this issue: Women as a special population—Hypertriglyceridemia during pregnancy
Ischemic heart disease in women | Plus—2010 Scientific Sessions Preview

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From the NLA President: Making the right connections

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In March this year, the NLA Strategic Planning Group will again meet to review our progress toward our 5-Year Strategic Plan, and chart new directions for our association. We'll report back to you on the results in the following issue.

When it comes to planning and goals, we should consider that 10 years ago, the Healthy People 2010 (HP2010) initiative was launched by the US government to promote targets for coronary heart disease (CHD) mortality reduction.¹ Risk factors for both primary and secondary prevention were identified, and if these goals for reducing CHD and stroke had been achieved, the result would have been a 20% decrease in age-adjusted mortality rates. There were targets for mean total cholesterol (199 mg/dL), and targets for smoking cessation, obesity, diabetes, hypertension and physical activity levels. These are all factors that we as clinicians address in our field of medicine.

So how did we do? A recent study undertaken in part by the US Centers for Disease Control (CDC) indicates that approximately 400,000 people in the US will die of CHD this year,² but had we achieved the HP2010 targets that

number would be trimmed in half. We clearly have our work cut out for us. A part of this will involve communicating our messages. Our members should take advantage of opportunities to educate the public, whether by giving presentations, publishing articles, books, letters to the editor, and of course teaching our own patients about ways they can reduce risk.

In addition to the work we do, there are related organizations who share our mission and it is vital that we seek out ways to collaborate with them, increasing our strength in numbers. As mentioned in this issue's news section we are collaborating with the American College of Cardiology in support of their Campaign for Patient Access, and the American Heart Association is co-sponsoring all 3 NLA meetings this year through their councils on Clinical Cardiology, Cardiovascular Nursing, and Nutrition, Physical Activity and Metabolism (NPAM). We have also strengthened our ties with the American Society for Preventive Cardiology, with whom we co-sponsored two symposia last year and we hope to join forces with them again in 2010. The Preventive Cardiovascular Nurses Association has long had members who are also on NLA

boards, and we are looking for additional collaborations that are a natural fit for us, such as the American Diabetes Association and the American Association of Cardiovascular and Pulmonary Rehabilitation (AACVPR).

We also need to communicate well among ourselves. When we share our knowledge with one another, we all improve. The NLA Scientific Sessions will be held in Chicago, May 13–16, and you can register online now at www.lipid.org. Attending NLA meetings helps you learn from leaders in our field, and also makes it easy to network with your colleagues and exchange information and ideas. In this issue of the *Lipid Spin*, we're giving you a peek inside the practice of one of our members, Dr. Rolando deGoma. This is the kind of practical information you can gain from interacting with other NLA members that

NLA President *(continued on page 30)*



Discuss this article at www.lipid.org

Go to "Topics" and look for "Making the right connections."

From the NELA President: Continuing to push forward



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NELA is pleased to present this issue of the Lipid Spin dealing with hypertriglyceridemia, experience with coronary calcium scoring, and two articles focusing on women. As one ages, and female classmates become postmenopausal, the announcement of the first female dying from a heart attack becomes an ominous warning that women indeed do die of coronary disease and the impersonal statistics take on more personal alarm. And premenopausal hyperchylomicronemic women with a past history of pancreatitis do wish to become pregnant even given the rather frightening statistics for mother and fetus. How should the clinical lipidologist respond, what advice would you give? Hopefully, this issue, if not giving absolute answers, will help you in considering your response before that rare woman first confronts you.

As the third-largest regional branch of the NLA, we in NELA are still trying to develop a bit more focus, as well as a warmer, friendlier group spirit that reaches beyond the active members on the Board. We've requested that NLA provide a telephone Internet meeting where a staff member can teach us better how to use and interact on the NLA site, rather than just passing through it on our way to the next 50 items on our daily agendas. We look forward to planning with SELA and then attending the Summer Clinical Update in Washington, DC, in late August.

Finally we are concerned about the calls for consultation from graduated fellows in cardiology and endocrinology with rather straightforward lipid interventions. The NELA Board has forwarded these concerns on to the NLA Graduate Medical Education (GME) Committee. The GME Committee is developing a basic clinical lipidology fellowship curriculum to offer training programs. Additionally, the NLA invites fellows and allied health trainees the opportunity to participate in any of the 3 Lipid Management Training Courses offered in 2010 at no charge along with a \$500 travel scholarship. The Committee

has also recently completed a web-based assessment exam for fellows on lipid metabolism and dyslipidemia management that is available now to Program Directors. Call the NLA office or contact fellows@lipid.org for more information about how to access any of these programs, and further details are given on page 31 of this issue of the *Lipid Spin*. ■

Clinical Feature: Severe hypertriglyceridemia

A case report and topic review

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Introduction

Our interest in this entity was stimulated by a patient with severe hypertriglyceridemia who was recently admitted to our hospital. We will present the case scenario and review hypertriglyceridemia, including its epidemiology, pathogenesis, clinical implications and treatment.

The case is a 27-year-old Hispanic male who presented with 2-day history of polyuria and polydipsia. Review of system was otherwise negative except for a new eruptive skin rash and a recent 30-pound weight gain. He was diagnosed with type 2 diabetes mellitus (DM) 3 years prior to admission complicated by diabetic ketoacidosis (DKA), hyperlipidemia and acute pancreatitis. He was treated with metformin and glyburide. He subsequently stopped these medications about 1 year prior to admission due to episodes of hypoglycemia. His family history was

positive for type 2 DM in his father, but negative for cardiovascular diseases or dyslipidemia. He drinks beer occasionally. His physical examination revealed a body mass index (BMI) of 39, lipemia retinalis and eruptive xanthomas mainly on the posterior chest wall and buttocks. His blood sample appeared to be lipemic (Fig 1). The laboratory data on admission showed a plasma glucose of 563 mg/dL, HbA1c=11.3%, triglyceride (TG) 16,040 mg/dL, total cholesterol



Figure 1. Lipemic plasma from the patient of the reported case (serum triglyceride 16,040 mg/dL)



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Go to "Topics" and look for "Severe hypertriglyceridemia."

(TC) 913 mg/dL and HDL-C 13 mg/dL. He had normal liver, renal and thyroid functions and a normal lipase level. The patient was admitted and treated with an insulin drip and IV fluids. He was also kept NPO (nothing by mouth). Twenty-four hours later heparin infusion at 600u/hr was added due to his persistent severe hypertriglyceridemia. His serum triglyceride gradually came down and his regimen was switched to PO (oral therapy) and subcutaneous medications on day 3. He was put on a low-fat American Diabetes Association (ADA) diet, fenofibrate 145 mg, niacin SR 1000 mg, and omega-3-

acid ethyl esters. His diabetes was well controlled with insulin glargine, metformin and glipizide. The patient's serum triglyceride had been reduced to 1777 mg/dL when he was discharged on day 10 and was 615mg/dL on outpatient follow-up 16 days after discharge. The trend of his triglyceride levels is shown in Fig. 2.

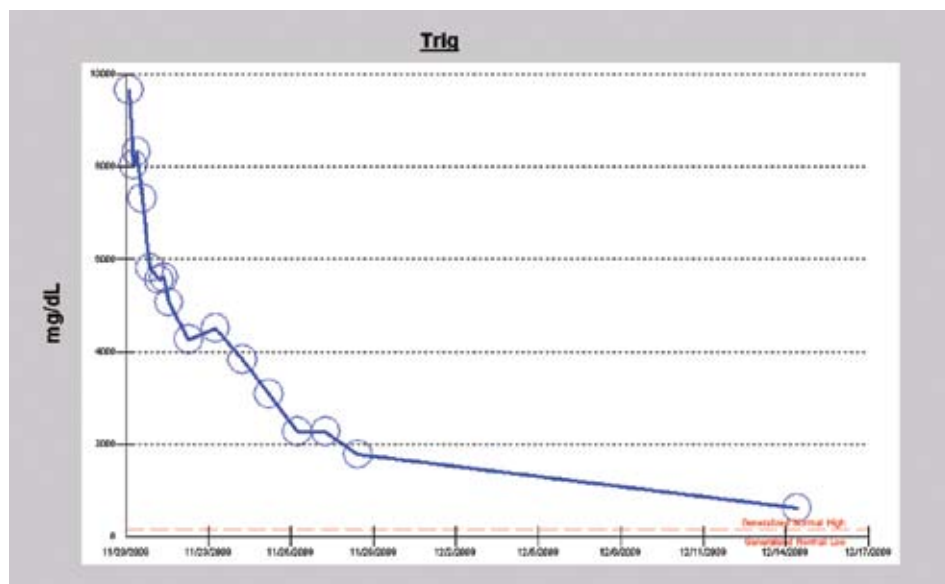


Figure 2. The trend of serum triglyceride change after treatment in the patient of the reported case.

Definition and prevalence of hypertriglyceridemia

Hypertriglyceridemia (HTG) is a common form of a lipid disorder. NCEP ATP III guidelines have suggested four classes of HTG according to the risk for cardiovascular disease: normal (<150 mg/dL), borderline high (155–199 mg/dL), high (200–499 mg/dL), and very high (>500 mg/dL). When TG is >1000mg/dL, the risk of developing acute pancreatitis becomes significantly increased and requires aggressive treatment.

The percentage of adults in the US with triglyceride levels above 150 mg/dL, 200 mg/dL, 500 mg/dL and 1000 mg/dL is 33, 18, 1.7, and 0.4 percent, respectively.¹ The incidence is highest (80–88%) in patients with premature coronary disease (CHD), which is about twice that of age-matched controls without CHD.^{2,3}

Pathogenesis and Classification

The two main sources of triglycerides are dietary fat and liver. TG are trafficked in all the lipoproteins, with the vast majority within chylomicrons (CM), which are formed in the small intestine, and very-low-density lipoproteins (VLDL), which are produced in the liver (Fig 3).

In capillaries within adipose and muscle tissue, these lipoproteins and CM are hydrolyzed by lipoprotein lipase (LPL) into free fatty acids. Apolipoprotein (Apo) C-II is a cofactor of LPL for chylomicrons degradation. Hypertriglyceridemia results from increased production from liver and/or intestine or through decrease peripheral catabolism.

Primary hypertriglyceridemia

Among the six familial hyperlipoproteinemias (Frederickson classification), only type IIa does not have HTG. Type I and V have severe HTG, and type IIb, III and IV usually have moderate HTG (Table 1).

Type I hyperlipotroteinemia (familial chylomicronemia syndrome) is defined by isolated severe chylomicronemia caused by LPL or ApoC-II deficiency due to gene mutations. Both of these deficiencies

have autosomal recessive inheritance and have a prevalence of approximately 1 in 1 million (even less in ApoC-II deficiency) in the population. Patients usually present in childhood with recurrent episodes of pancreatitis, lipemia retinalis, eruptive xanthomas and hepatosplenomegaly. The fasting triglyceride levels are almost invariably >1000 mg/dL.

Type V hyperlipotroteinemia (mixed hypertriglyceridemia) is characterized by elevated CM and VLDL, which cause severe HTG and moderately elevated cholesterol. It is an autosomal dominant disorder of unknown etiology. Its clinical presentation is similar to Type I hyperlipotroteinemia, but usually present in adulthood and patients often have secondary risk factors for HTG. It can be diagnosed by confirming the presence of CM and excess VLDL on agarose gel electrophoresis or ultracentrifugal analysis.

Type IV hyperlipotroteinemia (familial hypertriglyceridemia) is defined by an isolated elevation of VLDL. It is a relatively common (~1 in 500) autosomal dominant disorder of unknown etiology characterized by moderated elevated plasma TG. It is frequently associated with metabolic syndrome and is associated with increased risk of cardiovascular diseases.

Type IIb hyperlipotroteinemia (familial combined hyperlipidemia) is also common in the general population (2–5%), with autosomal dominant inheritance and unknown molecular defects. It is characterized by moderately elevated TG and more prominent elevated TC and LDL-C. Patients are at significant increased risk for cardiovascular diseases.

Finally, type III hyperlipotroteinemia (familial dysbetalipoproteinemia) is also characterized by mixed hyperlipidemia due to the accumulation of CM and VLDL remnants. It is due to genetic variations in ApoE that interfere with its ability to bind lipoprotein receptors.

| phenotype | I | IIb | III | IV | V |
|--------------------------|-----------------|--------------|-------------------------------|--------------------|----------------------|
| Lipoprotein, elevated | chylomicrons | LDL and VLDL | chylomicron and VLDL remnants | VLDL | chylomicron and VLDL |
| Triglyceride | ↑ ↑ ↑ ↑ | ↑ ↑ | ↑ ↑ to ↑ ↑ ↑ | ↑ ↑ | ↑ ↑ ↑ ↑ |
| Cholesterol | ↑ to ↑ ↑ | ↑ ↑ to ↑ ↑ ↑ | ↑ ↑ to ↑ ↑ ↑ | - to ↑ | ↑ ↑ to ↑ ↑ ↑ |
| Xanthomas | Eruptive | - | Palmar, tuberoeruptive | - | eruptive |
| Pancreatitis | +++ | - | - | - | +++ |
| Coronary atherosclerosis | - | +++ | +++ | +/- | +/- |
| Molecular defects | LPL and apoC-II | unknown | ApoE | ApoA-V and unknown | ApoA-V and unknown |

Table 1. Primary hypertriglyceridemia (Frederickson classification)

The most common defect is an Apo-E2 substituted for the usual apo-E3. It is rare in general population (1–2 in 20,000). Affected people usually have mixed hyperlipidemia with low LDL-C. They often have characteristic tuberoeruptive and palmar xanthomas. Due to the increased cardiovascular risk, affected patients need aggressive treatment for their dyslipidemia.

Secondary hypertriglyceridemia

Several metabolic and lifestyle risk factors, medical diseases and medications raise the plasma triglyceride level (Table 2). These factors may be particularly important in patients with underlying defects in

triglyceride metabolism.

Diet and alcohol: A diet with positive energy-intake balance, high fat or high glycemic index content (>60% of energy) can contribute to HTG. Although regular alcohol use can raise plasma HDL-C, it is an important risk factor for HTG by stimulating liver secretion of VLDL and impairing lipolysis.

Obesity, diabetes and the metabolic syndrome are the most frequently associated risk factors for secondary HTG. The high insulin level and insulin resistance that underline these conditions have multiple effects on lipid metabolism:

They (1) decrease LPL activity; (2) increase release of free fatty acids from adipose tissue, and; (3) increase fatty acid synthesis and VLDL production. Diabetic ketoacidosis is frequently accompanied by HTG due to an increased hepatic influx of free fatty acids from adipose tissue.

Other medical conditions including hypothyroidism, renal and liver diseases are also associated with various degrees of hypertriglyceridemia. Hypothyroidism can cause mild hypertriglyceridemia and moderately elevated LDL-C. Nephrotic syndrome is often associated with pronounced mixed hyperlipidemia, and mild HTG is often seen in ESRD patients. Hepatitis due to infection, drugs or alcohol is often associated with increased VLDL synthesis and mild to moderate HTG. However severe hepatitis and liver failure are often associated with dramatic reduction in plasma triglycerides due to reduced lipoprotein biosynthetic capacity.

Medications that can cause HTG are listed in Table 2, on the following page.

Clinical implications

Acute pancreatitis

The most important complication of severe HTG is acute pancreatitis (HTGP), which is a very serious and potentially life-threatening disease. HTG accounts for 1–10% of all cases of pancreatitis and even up to 50% in pregnant women with acute pancreatitis.^{4,5} Hydrolysis of triglycerides

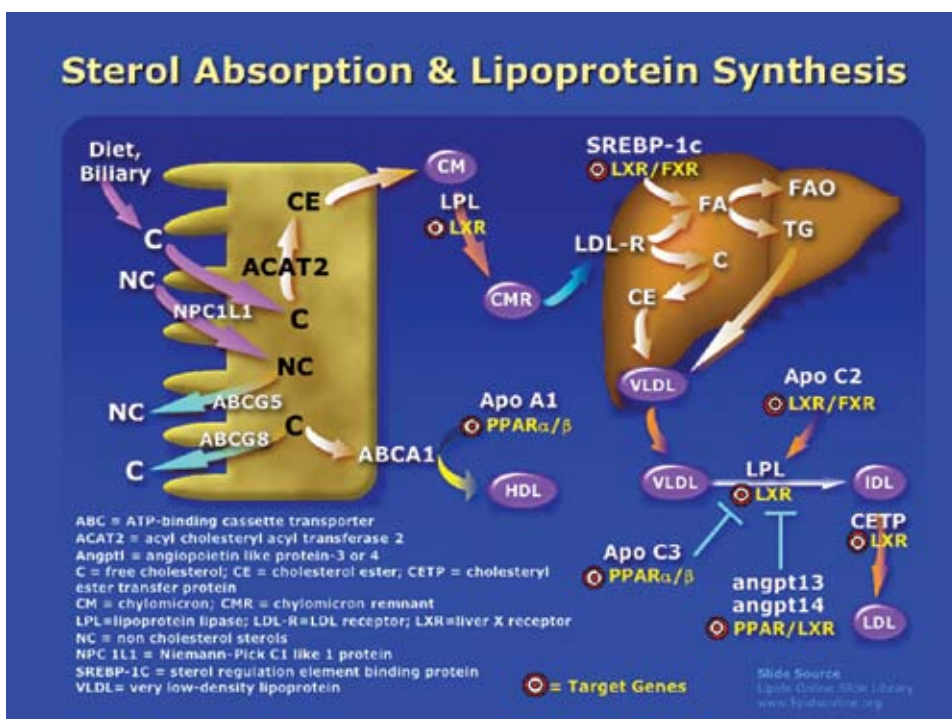


Figure 3. Sterol absorption and lipoprotein synthesis © Wolters Kluwer Health, used with permission.

Lifestyle

A diet with a positive energy-intake balance and a high fat or high glycemic index content
Alcohol consumption
Insufficient physical activity
Obesity

Diseases

Metabolic syndrome
Type 2 Diabetes mellitus, diabetic ketoacidosis
Nephritic syndrome and ESRD
Hypothyroidism
Liver diseases

Medications

Corticosteroids
Estrogen, Tamoxifen
Antihypertensives: nonselective β -blockers, thiazides
Isotretinoin
Bile-acid-binding resins
Cyclophosphamide
Antiretroviral regimens: especially for HIV infections
Psychotropic medications: phenothiazines, second-generation antipsychotics

Cardiovascular Munster (PROCAM) study demonstrated that moderate increases in triglycerides are associated with an increased risk for CHD independently. A 6-fold increased CHD risk was observed in patients with triglycerides >200 mg/dL and LDL-C/HDL-C >5.⁹ A meta-analysis of 17 population-based studies identified a 32% increase in CHD risk in men and 76% in women associated with triglyceride elevation of 1 mmol/L.¹⁰ Another meta-analysis of 29 Western prospective studies involving a total of 10,158 incident CHD cases from 262,525 participants found

Table 2. Secondary causes of and contributors to hypertriglyceridemia

by pancreatic lipase and the formation of free fatty acids that induce inflammatory changes are postulated to account for the development of HTGP, yet the exact pathophysiology remains unclear.⁴

A few patients can develop pancreatitis when their fasting TG is 500–1000 mg/dL, however this risk becomes more clinically significant when fasting TG exceed 1000 mg/dL. In the study by Fortson et al., the average serum triglyceride concentration at presentation of pancreatitis was 4587 mg/dL.⁵ However, the threshold for developing pancreatitis can vary widely. A recent study observed 129 patients with TG >1000 mg/dL and found that only 20% had pancreatitis.⁶

The clinical features of patients with HTGP are generally not different from patients with acute pancreatitis from other causes, and there is some evidence that HTGP is associated with a higher severity or a higher complication rate.^{6,7} However, there is no correlation between the severity of pancreatitis and the degree of HTG.

Cardiovascular diseases

Elevated serum triglyceride concentrations are associated with an increased risk for CHD, directly as well as indirectly, because more often such elevations are associated with other risk factors for atherosclerosis. It is controversial how much isolated HTG is associated with CHD and if treating isolated HTG would prevent CHD. However, when HTG is identified, a thorough investigation for the metabolic syndrome is recommended.

HTG is recognized as an independent risk factor for CHD. This independence suggests that some triglyceride-rich lipoproteins, especially remnant lipoproteins (VLDL and IDL), are atherogenic. They are cholesterol-enriched particles and have many of the properties of LDL. For these reasons, in all patients with elevated triglyceride levels, elevated remnant lipoproteins should be a target of therapy.⁸ Many studies have shown that elevated plasma TG concentrations are independently associated with increased risk for CHD. The Prospective

that HTG had a significant increased risk for CAD.¹¹

Many people with elevated triglycerides are at increased risk for CHD, but sometimes this increased risk cannot be independently explained by triglycerides. HTG is frequently associated with other metabolic and biochemical abnormalities that are linked to atherosclerosis. These include type 2 DM, obesity, increased LDL, decreased HDL, hypertension, increased plasma viscosity, increased inflammatory molecules, and prothrombosis. The finding of elevated serum triglycerides should prompt investigations for identifying the persons who are at risk and who need intervention for risk reduction. The conclusions of the MELANY study corroborate triglycerides as a sensitive marker of lifestyle changes in healthy young males. A decrease in initially elevated triglyceride levels is associated with a dramatic decrease in CHD risk compared with stable high triglyceride levels.¹²

Acute treatments for severe HTG

If patients with severe HTG are admitted for acute pancreatitis or other reasons (such as DKA), aggressive treatments for lowering plasma triglyceride level to below 1000 mg/dL (or even <500 mg/dL) are needed to treat or prevent acute pancreatitis. Intravenous (IV) insulin is the first line treatment. Heparin or apheresis could also be considered, along with diet intervention and oral antihyperlipidemia agents.

Insulin activates LPL and leads to acceleration of CM and VLDL degradation. Since most patients with HTG are obese and insulin resistant, substantial amounts

to remove TG from plasma. However, it is invasive and expensive and usually reserved for severe HGTP that does not respond to other treatments. However, a small case-control study failed to show any benefit of apheresis on mortality or complications of HTGP.⁷ The role of apheresis in secondary prevention of pancreatitis in patients with severe HTG lacks clinical evidence, although individual experience has been reported.^{14,15}

General nonpharmacological treatment

Patients with hypertriglyceridemia should avoid known risk factors (see above secondary hypertriglyceridemia). Low fat diets and the avoidance of

observed in women.¹⁷ Another recent study showed that plasma triglyceride response to diet and weight loss is about 25%, with marked variation among patients.¹⁸

Oral pharmacological agents

Fibrates: Fibric acid derivatives such as gemfibrozil and fenofibrate are first line pharmacological therapy for hypertriglyceridemia. They lower serum triglycerides by 18 to 45 percent and raise serum HDL-C by 15 to 25 percent. Two factors contribute to the fibrate-induced fall in triglycerides: Reduced hepatic secretion of VLDL and facilitated clearance of triglyceride-enriched lipoproteins by the stimulation of LPL activity. These effects

| Drug class | Decrease in TG (%) | Regimen | Side effects | Effect on LDL-C | Effect on HDL-C |
|-------------------------|--------------------|---|---|-----------------|-----------------|
| Fibrates | 18–45 | Gemfibrozil, 600 mg twice a day Fenofibrate 145 mg daily | Myositis, cholelithiasis | ↓ | ↑ |
| Nicotinic acid (Niacin) | 17–26% | 1500–2000 mg daily | Flushing, pruritus, nausea, hepatitis, activation of migraine | ↓ | ↑ |
| Omega-3 fatty acid | 19–44 | Lovaza 4g daily | May increase LDL-C, ALT and prolong bleeding time | ↑ | – |

Table 3. Oral Pharmacologic agents for hypertriglyceridemia

of IV insulin are needed to lower TG level quickly. It is very useful in patients with poorly controlled diabetes, but is also used in non-diabetic patients along with IV dextrose.

Heparin stimulates endothelial release of LPL, its effect on lowering TG has been evaluated in several studies but the results remain controversial.¹³ This effect may be related to its transient raising of LPL followed by the increased degradation and depletion of plasma LPL stores resulting in LPL deficiency. It's usually used as a second line therapy and combines with insulin drip if insulin alone is not sufficient.

Apheresis is a quick and effective way

high carbohydrates diets, decreasing or eliminating alcohol consumption, weight loss for obese patients, exercise, strict glycemic control in diabetic patients, and appropriate treatment for related medical conditions and avoidance of certain medications should be the first line therapy. For severe HTG, dietary fat <15-20g/day is usually recommended. Median-chain triglyceride (MCT) has also been used to lower plasma triglyceride levels effectively by reducing postprandial CM production, but the concern with MCT is its adverse effect on cholesterol.¹⁶ An early study showed that serum triglycerides fell by 8% with hypocaloric NCEP diet alone, and by 33% with diet and exercise in obese men; however, the above effect was not

are mediated at least in part by activation of peroxisome proliferator-activated receptors (PPARs) (see Table 3).

The effectiveness of fibrates in reducing cardiovascular disease outcomes has been controversial. A meta-analysis of 10 randomized trials (36,489 patients; both primary and secondary prevention trials) found that the long-term use of fibrates significantly reduces the occurrence of nonfatal MI but has no significant effect on other adverse cardiovascular outcomes.¹⁹

Nicotinic acid (Niacin) reduces triglycerides and cholesterol by inhibiting the hepatic production of VLDL and consequently its metabolite LDL. It also

Clinical Feature (continued on page 28)


Introducing a radio host
who gives you
the skinny
on fatty acids.



Dr. Brown

Alan S. Brown, MD
Director, Midwest Heart Specialists Prevention Center

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Practical Pearls: Hypertriglyceridemia during pregnancy

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Is there a genetic basis for hypertriglyceridemia in pregnancy?

In all likelihood, there is a strong genetic basis for the hyperchylomicronemia that can cause acute pancreatitis in pregnancy. Fundamentally, this disorder stems primarily from an inherited defect in the catabolism of chylomicrons due to diminished or defective lipoprotein lipase activity. This results in reduced catalytic removal of their diet-derived triglyceride cargo. Numerous individual genetic mutations of the lipoprotein gene and other genetic mutations that include apolipoprotein CIII overexpression, apolipoprotein CII deficiency, abnormal apoE isoforms and other catabolic defects. Any of these biochemical defects can inhibit catabolism of chylomicron triglycerides, and result in this potentially life-threatening event.

What strategies are available to prevent or abort an impending episode of

the above mentioned form of acute pancreatitis?

In evaluation of a female patient who is pregnant or about to become pregnant who has a history of abdominal pain after ingestion of fatty meals or has a prior fetal demise due to this condition, the evaluating physician should perform a detailed history and physical examination. The patient should be provided with strict dietary advice and recommendations for follow up. Frequent visits should be planned to monitor dietary and other lifestyle measures. Detailed dietary instruction should be given to restrict intake of dietary fat and avoidance of alcohol intake by the patient at all times. This preventive care requires close communication among the physician, the patient and a registered dietician and other specialists and allied health care specialist when indicated. Furthermore, this involves frequent measurement of serum lipids and visual examination of



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Go to "Topics" and look for "Hypertriglyceridemia during pregnancy."

serum/plasma specimens for turbidity and layering of triglycerides, and a review of this material with the patient. Episodes of abdominal pain due to dietary indiscretion may necessitate a brief hospital stay for rehydration and other therapy. The use of plasma triglyceride-lowering agents is usually also required. As the gestation progresses it may be necessary to modify the dosages of these agents and to monitor their effects.

Though severe hypertriglyceridemia in pregnancy is rarely encountered, its occurrence can result in bouts of abdominal pain due to episodes of acute pancreatitis that often result from dietary

Practical Pearls (continued on page 28)

Cardiology Corner: Ischemic heart disease in women



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Go to "Topics" and look for "Ischemic heart disease in women."

I first met Adelle in 1991, when she presented with effort-induced angina. She was 50 years old and had no cardiovascular risk factors. Her lipids were total cholesterol 172 mg/dL, triglycerides 82 mg/dL, high density lipoprotein cholesterol (HDL-C) 79 mg/dL, and low density lipoprotein cholesterol (LDL-C) 76 mg/dL. Her cardiac catheterization was normal. In 1999, because of ongoing symptoms, she had an exercise test, which revealed an excellent exercise capacity, reduplication of her symptoms, ischemic changes on EKG but a normal perfusion scan. In 2004 she presented to the ER with symptoms of food poisoning. She had an NSTEMI and a repeat cardiac catheterization was normal. In 2006 she had a repeat NSTEMI. Her repeat cardiac catheterization showed minimal atherosclerosis. Her

coronary calcium score was zero. She has subsequently developed lymphocytic vasculitis and Raynaud's phenomena. In spite of angiotensin converting enzyme (ACE) inhibitors, statins, calcium channel blockers, ASA, and L arginine, all aimed at improving endothelial function, she has persistent class 2 symptoms of angina. Her most recent lipids are cholesterol 146 mg/dL, triglycerides 41 mg/dL, HDL-C 98 mg/dL, and LDL-C 40 mg/dL.

The general public is unaware that cardiovascular disease is the leading cause of death in women, which is true for all racial and ethnic groups. Breast cancer is on the mind of all women, yet the age-adjusted mortality for cardiovascular disease is 3–5 times higher. Only 9% of primary care physicians, 13% of obstetricians and 17% of cardiologists were aware that more women than men die every year from cardiovascular disease. Over the past two decades, the age-adjusted mortality from cardiovascular disease has decreased more for men than women. This may be related to the fact that women with an acute myocardial infarction treated similar to men have a higher mortality rate than the men. In addition, they receive less evidence-based

treatment for coronary artery disease (CAD).

Traditional risk factors and the Framingham risk score underestimate risk in women for cardiovascular disease. There are also some gender-specific risk factors: polycystic ovary syndrome, pregnancy-induced hypertension and diabetes, markers of inflammation, metabolic syndrome, and collagen vascular disease are harbingers of premature cardiovascular disease in women.

Angina, described by Heberden in 1768, has many etiologies such as atherosclerosis, cardiomyopathy, and valvular heart disease. Angina is a clinical diagnosis and requires a detailed history. At times, women may present with anginal pain, which is atypical. It may not always be related to exertion, may be prolonged in duration and not promptly relieved by nitroglycerin.

Women are more likely than men to experience myocardial ischemia due to microvascular dysfunction. This may be related to either endothelial dependent (abnormal response to acetylcholine) or endothelial independent dysfunction (abnormal response to adenosine).

Cardiology Corner (continued on page 30)

Member Spotlight: A personal and professional journey

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Note: We have been hearing good things about Dr. Rolando deGoma's NJ Preventive Cardiology and Cholesterol Clinic, and he was kind enough to give an interview with the *Lipid Spin*. This is the story of how his practice has adopted many of the hallmarks of a successful lipid clinic. Those seeking to incorporate more lipid-focused treatment and counseling concepts into their work will undoubtedly find much to consider here. —ed

In the beginning

In private practice for over 25 years, Dr. deGoma was focused primarily on the diagnosis and treatment of heart disease, and many of his patients were in secondary prevention. As a result, he was treating patients' lipids, high blood pressure, and lifestyle risk factors such as smoking.

When the results of the Lipid Treatment Assessment Program (L-TAP) were published in February 2000, Dr. deGoma took careful note on the findings which compiled surveys of primary care doctors, noting how many are complying with NCEP guidelines. These were high statin prescribers, so it was surprising to see how few were getting their patients to goal (only 18% of patients with coronary heart disease were being treated to the target

of less than 100 mg/dL, and 72% were either not treated or treated inadequately). At about the same time, Dr. deGoma's cardiology group participated in similar retrospective compliance study expecting to see better performance. Much to his surprise, cardiologists did not significantly do a better job. It was at this point that Dr. deGoma decided to pursue a greater knowledge of lipidology. "I started to look around, wondering how I could do a better job. If there is a drug that reduces event rates by nearly half, that is safe and nearly 95% of patients can take, that is covered by

**"I could do more,
but not less"**

insurance and even has guidelines telling me which patients to treat, and I thought I was treating them but wasn't, there is something wrong here. But in 2001 there was little support for prevention," he recalls.

Fortunately, Dr. deGoma was able to obtain informal preceptorships at several lipid clinics, spending a few days at both



Discuss this article at www.lipid.org

Go to "Topics" and look for "Member Spotlight – Rolando deGoma, MD."

Dr. Daniel Rader's and Dr. Paul Ziajka's clinics. These were profound influences on Dr. deGoma and these well known experts had a major influence on him. In addition, Dr. Ziajka introduced Dr. deGoma to the Southeast Lipid Association (this was in the pre-NLA days). At this point, Dr. deGoma began to seek ways to incorporate lipidology as a form of preventive cardiology in his practice.

Building up a practice based on risk reduction

In his studies, it became apparent to Dr. deGoma that *compliance* was a key issue for both primary care physicians and cardiologists, who weren't getting many patients to goal, and for patients, who often do not adhere to their recommended treatment plan. "I began to audit my own practice," he says, "taking just 5 minutes at the end of every day to review my cases and check my own progress." Later, at a

SELA meeting, a member of the NCEP ATP III expert panel pointed out that the ATP III guidelines were only ‘mere suggestions,’ which surprised Dr. deGoma. “I decided to elevate the ATP III to my minimum standard of care. I can do more, but not less.”

Determined to go beyond the guidelines and press for powerful levels of risk



Dr. deGoma counsels a patient regarding risk.

reduction, Dr. deGoma began to analyze his patient base and his opportunities for intervention. He witnessed how coronary heart disease (CHD) impacted patients’ lives and families, how many patients in a cardiology practice seem to be in a revolving door of tests, procedures and surgeries. Deciding to reformat the guidelines in a way that he could implement them sensibly into his practice, Dr. deGoma broke them down into steps that could be implemented on first, second, third and subsequent visits. “A lipid management plan should be part of every routine cardiology visit, but it also needs to be implemented in a cost-effective manner. Every patient encounter is an opportunity for prevention,” he says. If a patient presents for any cardiac evaluation, a formal cardiac risk assessment is incorporated into the visit and a treatment plan enacted, with modification if necessary on follow-up visits.

“We know that lipid medication is very safe,” Dr. deGoma says, “so I decided to start with fairly high doses of statins when medication was indicated. The mean effects of each dose of statin on each lipid parameter are known. One of the problems seen in the L-TAP study was that many patients were not at goal because they were still on their initial starting statin dose 2 years later. The initial dose of a

selected statin should get the majority of patients to their LDL-C goal. An LDL-C goal below 100 mg/dL does not mean that the goal is 99. Ninety-nine is acceptable but 69 is better.” As a result, his patients began to reach and exceed their lipid level goals at impressive rates. This wound up to have disadvantages. “I was the biggest supporter of intervention in my group before. Our group’s interventional cardiologist eventually left our practice after 3 years because we were referring fewer and fewer patients to him.” Dr. deGoma published and presented clinical data from his practice at the 29th Society of General Internal Medicine (SGIM) Annual Meeting. Unlike the results of L-TAP, in deGoma’s practice some 85% of high-risk patients were treated successfully to LDL cholesterol below 100 mg/dL. Not only were just 15% not at goal, but he had more patients with LDL-C below 50 mg/dL than over 130 mg/dL.

Bringing patients into the treatment team

During follow up visits, all of Dr. deGoma’s patients end up in his consultation room after the exam room for discussion and patient mentoring. “I say to every patient after my examination, ‘Get dressed and we will talk in my office.’ Patient mentoring is incorporated in my website to provide a visual tool that makes it easier for my patients to grasp what I am explaining,” he says, “Physicians are not just healers, but also mentors and motivators.” He uses his consultation time based on the needs of the patient, explaining treatment strategies, answering questions, and working with the patient to set goals. “For secondary prevention patient, I might show a 4S trial slide, and for primary prevention, a AFCAPS/TexCAP or JUPITER trial slide” he says, “showing the magnitude of risk reduction—42% reduction in fatal heart attack, 37% reduction in need of future heart bypass, 30% reduction in stroke in the 4S or showing the early onset of benefits in the ASCOT trial, or early termination of the JUPITER trial after only 19 months due to benefits.” There’s more: “Patients might ask me what is LDL, they might have a misconception. I can show them cholesterol in the arterial wall or my YouTube video. I can show them what plaque looks like on intravascular imaging, and walk them through concepts like oxidation, progression and plaque rupture.” This may sound too time-consuming for a busy practitioner, but this approach is a key part of Dr. deGoma’s impressive results. By showing patients how primary prevention is better than secondary, and how optimal lipid therapy breaks the recurring cycle of events in secondary prevention and showing the risk reduction achieved by various measures, he gets them to understand their role in reducing their CHD risk.

“You have to be effective with your time,”

Member Spotlight (continued on page 29)

Committee and Awards Nominations

Honors and Awards

The NLA Honors and Awards Committee issues the NLA Distinguished Achievement Award, the Honorary Lifetime Member Award, and Fellow of the NLA designation. We always look to recognize and applaud member commitment to medicine, research, and the very highest levels of patient care.

Nominations for the following awards are open for 2010 through April 1, 2010

NLA Distinguished Achievement Award

This award is the highest honor conferred by the NLA. Candidates must be widely known for a major contribution to clinical lipidology (research, teaching, publishing, or service), whether as a single accomplishment or by a lengthy career.

Fellow of the NLA

Fellowship is reserved for NLA members who have made significant regional and/or national contributions to the science and practice of clinical lipidology. Fellows of the NLA are entitled to append their names with the designation, "FNLA."

Complete details about these awards, nomination criteria, and an online application form are available at: www.lipid.org/awards

Committee Service Opportunities

The success of the National Lipid Association depends on the spirit of volunteerism of the membership. Nominations are being gathered now for the 2010–2011 committees and for service on the national and regional chapter boards. Members are asked submit their

interest for positions prior to **March 15, 2010**. If you are looking to work more directly with the NLA, this is an excellent opportunity to support the association in its mission. The following committees will have openings and we are calling for volunteers:

Communications Committee

Open to: Members who wish to edit major NLA communications programs, such as the Lipid Spin and Website (lipid.org); other members shall form an Editorial Board. The committee reviews scientific content of NLA publications, plans communication strategy to NLA members, and considers standards for advertising in NLA publications.

Consumer Affairs Committee

Open to: Members with expertise in clinical medicine, public health, consumer affairs, dietetics, pharmacy and related legal issues. The Committee evaluates NLA advisory statements to the public, and makes recommendations regarding OTC products.

Membership Committee

Open to: Up to eight members (at least one from each regional chapter) appointed by the President and approved by the Board of Directors. Reviews applications

for membership to NLA and develops recruitment strategies and campaigns to acquire and retain members.

NLA CME Committee

Open to: Up to 12 members representing each regional chapter as well as Family Practice and Osteopathy. The Committee oversees all continuing medical education activities consistent with ACCME and AMA guidelines.

For complete details regarding these committees and their functions, visit: www.lipid.org/about/committees.

To apply for a committee assignment or to nominate a member, complete and fax the following form to: **904-998-0855**, or e-mail abeamer@lipid.org.

Interested in Board Position or Committee

Board or Committee: _____

Name of Nominee: _____

Title: _____

Address: _____

City: State: Zip: _____

E-mail: _____

Phone: _____ Fax: _____

Nominated By

Name of Nominator: _____

E-mail: _____

Phone: _____ Fax: _____

Suggested Supporting Documents

* Statement/Letter of Support – including bio/CV

* And any supporting letters/qualifications

Lipid Luminations: Accelerated progression of subclinical atherosclerosis—the lipid connection



C. MICHAEL WRIGHT, MD

The Cardiovascular Specialists
Hyannis, MA

Diplomate, American Board of Clinical Lipidology



Discuss this article at www.lipid.org

Go to "Topics" and look for "Accelerated progression."

Coronary artery disease (CAD) usually becomes symptomatic after the fifth decade of life. Coronary atherosclerosis begins much earlier, in the second and third decades of life. The progression of coronary atherosclerosis is dependent upon the long-term effects of various risk factors on the coronary artery endothelial, intimal, and medial layers.

Coronary artery calcium (CAC), as measured by ultrafast CT, is a marker for coronary atherosclerosis. The measurement of CAC in an asymptomatic population can be used to detect and quantify subclinical coronary atherosclerosis. I would like to briefly describe a paper¹ we recently published that demonstrates the usefulness of CAC in elucidating the natural history of CAD. In this particular instance, we were asking which of the classic modifiable risk factors present in middle age would be most

closely associated with CAC approximately three decades later.

The patients were participants in the Rancho Bernardo Study, a community-based epidemiologic study initiated in 1972–74. Enrollees were between the ages of 30 and 79, Caucasian, and middle or upper-middle class. Baseline assessments included cigarette smoking status, height, weight, blood pressure, fasting glucose, cholesterol and triglyceride levels.

Between 2000 and 2002, 422 of the surviving original participants with no history of CAD underwent CAC scoring at the LifeScore Clinic, which I founded in 1999. The mean age at enrollment was 41.8 years. The mean age at the time of CAC measurement was 69.4 years. In a previous paper from the LifeScore Clinic,² we showed that, for our population, at ages less than 45, median calcium scores are negligible, regardless of gender. By age 65–74, the median scores for men and women are, respectively, 180 and 20.

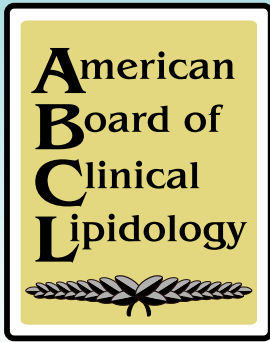
The absence of calcium on coronary CT has a 99.9% negative predictive value and equates to an annual event rate of 0.027%.³ Since atherosclerosis begins in

our teens and twenties, Atherosclerotic lesions with no CT-detectable calcium appear to represent a stage of the disease with less risk for the development of unstable plaque. As CAC score increases, the risk for adverse coronary events goes up dramatically. Even a CAC score of 1–10, still considered a sign of minimal plaque, confers a 3-fold increase in risk.⁴

After age and gender, the only statistically significant variables predicting higher CAC scores in the Rancho Bernardo survivors were body mass index (BMI), total cholesterol (TC) and triglycerides (TG). The mean TG level in those with minimal CAC (score 0–10) was 79 mg/dL, vs. 99 mg/dL in those with severe CAC (score >400). The mean TC in the minimal CAC group was 183 mg/dL vs. 201 mg/dL in the severe CAC group. The mean BMI measurements were 23 kg/m² and 24.3 kg/m², respectively.

This study provides insight into the natural history of CAD. Middle-aged men and women can be identified who will have accelerated rates of coronary plaque formation. These accelerated rates appear to be dependent upon the presence of

Lipid Luminations (continued on page 30)



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"I found as a certified lipidologist that I gain more professional credibility among my peers both locally and nationally. Even after the examination, my knowledge has continued to grow substantially from the excellent educational materials from the Journal of Clinical Lipidology and NLA CME opportunities. Nothing else comes close in comparison."

*J. Ross Tanner, DO, FACP
Diplomate, American Boards of Internal Medicine and Lipidology
Alaska*

For an application, handbook, eligibility requirements, and examination information, visit the ABCL website at:

www.lipidboard.org

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**2010 EXAMINATIONS:
NEW IN 2010 - Exam is Now Offered in Computerized
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Spring Testing Window
March 15–April 15, 2010
(Application Deadline: February 26, 2010)

Summer Testing Window
June 14–July 14, 2010
(Application Deadline: May 17, 2010)

Fall Testing Window
September 20–October 20, 2010
(Application Deadline: August 30, 2010)

Case Studies: From the files



THOMAS DAYSPRING, MD, FACP, FNLA, NCMP

Clinical Assistant Professor of Medicine
New Jersey Medical School

Director, North Jersey Institute of Menopausal Lipidology
Wayne, NJ

Diplomate, American Board of Clinical Lipidology



We encourage you to discuss this case and others with Dr. Dayspring at www.lipid.org

Cases are now posted on the NLA Community page under the "Lipidaholics" group.

This article focuses on actual lipid disorders from actual patients, and how one clinical lipidologist evaluates the available clinical and laboratory data and then arrives at a therapeutic solution. Dr. Dayspring has been in practice 33 years and lectures extensively through the country on lipids and lipoprotein disorders.

CASE 1: A Negative Calcium Score Presents a Mystery

I was asked about a 61-year-old Caucasian male with no cardiovascular symptoms or history other than having mild hypertension controlled with atenolol 25 mg daily. He has known about his low high-density lipoprotein cholesterol (HDL-C) for 30 years. He actively and regularly exercises. An Ultrafast CT was negative

for calcium. There are no first-degree relatives with coronary heart disease (CHD) but a grandmother died at age 69 of an myocardial infarction (MI). His height is 5'9" and weight is 152 pounds. Current BP is 130/86.

A laboratory workup revealed:

FBS = 84
hs-CRP = 0.033
Lipid Profile:
TC = 146 mg/dL
LDL-C = 119 mg/dL
HDL-C = 22 mg/dL
TG = 36 mg/dL
Total LDL-P 1801 nmol/L (high risk)
LDL size is 20.1 nm: small (Pattern B)
Large HDL-P = 1 umol/L (an almost total absence of large HDL particles)
Small HDL-P = 20 umol/L (reduced)
Large VLDL = 0.5 nmol/L (perfect)

Analysis

The question is why does this person have a negative coronary calcium score with such a serious elevation of LDL-P and a lifetime of very low HDL-C? Well, a negative calcium score does not mean atherosclerosis is not present. A zero

calcium score is not 100% reassuring. Calcium scoring can miss soft plaques, which play an important role in acute coronary syndromes.¹ So because of the lipid/lipoprotein abnormalities we have to take this patient seriously.

Let's start the analysis as if we had only the lipid profile but no lipoprotein measurements. Using the National Cholesterol Education Program Adult Treatment Panel III (NCEP ATP-III) guidelines (2004 addendum), he has 3 major risk factors: age, hypertension, and HDL-C < 40 mg/dL. He would qualify for Framingham Risk Scoring and has a 10-year-risk of 16%, putting him in the moderately-high-risk category (≥ 2 risk factors with FRC of 10–20%). He only has 2 (hypertension and low HDL-C) of the 5 criteria for the metabolic syndrome and a normal hs-CRP. His LDL-C goal of therapy would be < 130 mg/dL, with an option for 100 mg/dL. Since he does not have hypertriglyceridemia, there is no non-HDL-C goal. The patient is therefore at the regular—but not at the optional—LDL-C goal of therapy. Although NCEP recognizes low HDL-C as a major independent predictor of risk, there is no specific HDL-C goal of therapy (astonishingly,

many providers are unaware of this fact). In addressing the treatment of low HDL-C patients, NCEP suggests that the LDL-C be brought to goal. Had TG been elevated, normalizing non-HDL-C would be the goal. Of course, to achieve that goal NCEP stresses therapeutic lifestyle changes (which this man seemingly does quite well) and, if needed, medication.

Since we do have lipoprotein measurements, let's leave the lipid era: It is lipoproteins, specifically apo-B containing lipoproteins, that traffic the sterols into the artery and risk directly correlates with particle number. The LDL-P in this man is quite abnormal with a level at the 80th percentile Framingham, and 90th percentile MESA population cutpoint. The LDL particle size is small, but we now know that, contrary to previous belief, LDL size is not an independent predictor of risk. The major use of LDL size is basically as a marker of insulin resistance.² Please note that the LDL-C elevation of 119 mg/dL is the 75th percentile MESA population cutpoint, and thus there is some discordance between LDL-C and LDL-P in

this case. Risk always follows LDL-P better than LDL-C.³

With respect to HDL particles, we know he lacks both large and small HDL particles, which of course anyone with an HDL-C of 22 mg/dL would. The number of large HDL particles drops dramatically with HDL-C values <40 mg/dL, and the number of small HDL particles drops at values <20 mg/dL.⁴ Epidemiological data has shown that a lack of large HDL-C is also an independent predictor of CV risk as well as a marker of insulin resistance (IR), but such patients usually have increased—not reduced—small HDL-P. Yet the perfect TG value, lack of large VLDL, glucose of 84 mg/dL, excellent BMI and lifestyle do not support IR. Thus we should be thinking that a genetic disorder of HDL, hypoalphalipoproteinemia, might be present. Do we have a disorder of apoA-I production, increased apoA-I catabolism (disappearing HDL syndrome), underlipidation of HDL or increased delipidation of HDL?

What are some of the potential diagnostic

possibilities?⁵ (1) Inability to produce enough Apo A-I, the HDL precursor protein (apoA-I and HDL-P are quite low). Usually the HDL-C is extremely low. (2)

“Speculating on genetic HDL disorders is great fun, but treatment always comes down to reducing atherogenic (apoB) particles, i.e., LDL-P”

ApoA-I Milano or other apoA-I structural abnormality (not associated with premature CHD). The HDL-C is usually lower than 22 mg/dL. (3) Heterozygous LCAT deficiency or Fish Eye Disease (not always associated with CHD). In such patients the NMR report will note the presence of lipoprotein x (not found in this case). The HDL-C is usually much lower. (4) Heterozygous reduction of ABCA1 (ATP binding cassette transporter A-I). This membrane protein transfers intracellular-free cholesterol to cholesterol-acceptor proteins like apoA-I or apoE. With a reduction in hepatic ABCA1, there will be underlipidation of immature HDL species and low HDL-C. Such patients have low HDL-C but are not necessarily at risk for CHD.⁶ Abnormalities of hepatic or endothelial lipase, and phospholipid transfer protein, or subtle combinations of the above can also dramatically affect HDL-C by enhancing HDL lipolysis (catabolism). Performing HDL mapping can help diagnose specific HDL disorders.

Speculating on genetic HDL disorders is



great fun, but treatment always comes down to reducing atherogenic (apoB) particles, i.e., LDL-P. The therapeutic mission in this patient is to normalize the LDL particle concentration.⁷ Step one, in a patient with a TG <500 mg/dL, is to prescribe a powerful statin. With the LDL-P so high, I would usually advise rosuvastatin (Crestor) starting at 20 mg. If the statin monotherapy did not reduce the LDL-P to goal (<1000 nmol/L) the easiest add-on therapy would be ezetimibe (Zetia) 10 mg. Keep in mind there is no specific NCEP, ADA/ACC or AACC⁷ HDL-C goal of therapy. Indeed, similar to the NCEP advice with lipid concentrations in persons with low HDL-C, the ADA/ACC consensus statement advises that in low HDL-C states, getting apoB to goal is the preferred strategy.⁸

With a negative calcium score and no insulin resistance, if I got LDL-P to goal I'd be content. However, there is emerging data showing that raising HDL-P might be beneficial. In a major analysis of statins and HDL-C and apoA-I, the HDL-C increase achieved by simvastatin, atorvastatin and rosuvastatin were independent of the LDL-C decrease, but baseline HDL-C and triglycerides and the presence of diabetes were good predictors of statin-induced increases in HDL-C. Extended-release niacin (Niaspan) at 2000 mg doses offer some additional apoB, e.g., LDL particle, lowering and might help raise HDL-P. Since the patient is not insulin resistant and the TG are perfect I am not sure that fenofibrate would do much.

CASE 2: A Delicate Balance

I saw a 67-year-old man with a history of hypercholesterolemia for over a decade. He had been treated with lifestyle and atorvastatin (Lipitor) 10 mg for many years. About a year and a half ago, he had chest pain and was found to have 2 vessel disease. He had a RCA stent

and 6 months later another stent in the LAD. The atorvastatin was increased to 40 and then 80 mg daily. He also takes 1000 mg of omega-3 (Lovaza), clopidogrel bisulfate (Plavix) and metoprolol as well as allopurinol for gout. His family history is extremely positive for CAD in that his mother died at 78 of an MI, his father had 3 MIs with the first at age 55 before dying at age 82, and a younger brother with 4 stents. He is normotensive and not obese.

Lipid profile on atorvastatin 80 mg:

TC = 124 mg/dL
HDL-C = 47 mg/dL
LDL-C = 57 mg/dL
TG = 120 mg/dL
Total LDL-P = 401 (well below goal)
LDL size large at 21.0
Total HDL-P = 39 (excellent)
Omega 3 index = 6.8% (desirable >8%)
Chemistry profile and vitamin D level are normal

monotherapy and the effect it often has sterol absorption. Cholesterol homeostasis requires a delicate balance between absorption and synthesis. Hepatocytes and enterocytes have great genetic powers to regulate both processes. People who genetically over absorb cholesterol usually have very little synthesis (are statin hyporesponders) and, conversely, overproducers of cholesterol under-absorb cholesterol (are statin hyper-responders). Complicating this process is that humans not only ingest cholesterol, but also substantial amounts of noncholesterol sterols (sterol molecules that are very similar in structure to cholesterol but with different ethyl-methyl side chains). Because they are more oxidizable, noncholesterol sterols have the potential to be significantly more atherogenic than is cholesterol. Unlike cholesterol, they serve no functions in humans. Enterocytes have proteins on their cell membranes that regulate sterol absorption. The Niemann Pick C1 Like 1 Protein (NPC1L1)



This man seems to be excellently controlled with his current regimen, although based on the Omega-3 index¹⁰ I increased his Lovaza to 2000 mg daily. My major concern was his high-dose statin

internalizes and sterol export transporters (called ATP binding cassette transporters G5 and G8) return noncholesterol sterols and unneeded cholesterol to the gut lumen after they are absorbed. These

membrane transporters also exist at the hepatobiliary interface. People who lack or have downregulated G5G8 sterol transporters have a very rare disease called sitosterolemia or phytosterolemia and experience very premature atherosclerosis.

There are laboratory assays available to evaluate cholesterol synthesis and absorption. In the 37 step cholesterol synthesis pathway, one of the final sterols is lathosterol which therefore serves as a marker of cholesterol synthesis. Both sitosterol and campesterol (plant sterols) serve as markers of intestinal absorption. Most humans absorb about 50% of the sterols that are in the gut lumen (the origin of the vast majority of the cholesterol is biliary not dietary or exogenous). Plasma was sent to the lab for a cholesterol balance test, which evaluates markers of absorption and synthesis. When looking at synthesis and absorption marker differences among two groups, one needs to be certain that these effects are independent of the cholesterol concentration and therefore the values are expressed relative to cholesterol (i.e., sterol/total cholesterol ratio). In this patient the Campesterol/C ratio was extremely high at 922 as was sitosterol/C ratio at 502. The Lathosterol/C ratio, a marker of synthesis was zero.

This patient has a dramatic reduction in cholesterol synthesis (likely because of the atorvastatin 80 mg) and dramatic over-absorption of sterols. However, we also cannot rule out that he has some form of sitosterolemia that has been worsened by the statin. Indeed the phytosterol absorption puts him at the level of a patient with sitosterolemia. Sterol hyperabsorption is a well known adverse side effect of statins, especially atorvastatin. In data from STELLAR, both atorvastatin and rosuvastatin (at maximum dose) inhibited lathosterol/C (marker of synthesis) the same (atorva 64% and

rosuva 68%). Both drugs also increased absorption: campesterol/C (rosuva 52% and atorva 72%), sitosterol/C (rosuva 67% and atorva 96%). If we average the two markers of absorption there seems to be a 25% difference. The authors state:

“We noted a significant difference between these two statins with rosuvastatin not raising the relative amounts of campesterol or sitosterol as much as atorvastatin. Therefore, rosuvastatin caused less of an up-regulation in markers of fractional

“Most humans absorb about 50% of the sterols that are in the gut lumen (the origin of the vast majority of the cholesterol is biliary not dietary or exogenous)”

cholesterol absorption than atorvastatin, indicating that this statin may have less of an effect on the intestine than atorvastatin. Thus with regard to the absorption markers, both statins have different pharmacokinetic properties, which may account for the somewhat greater efficacy in LDL-C lowering and HDL-C increasing for rosuvastatin than atorvastatin.”¹¹

By markedly inhibiting cholesterol synthesis (revealed by the very low lathosterol level) sterol homeostatic mechanisms induce an upregulation of the proteins (NPC1L1) that increase sterol absorption from the gut and from the bile back into the liver and induce a downregulation of ABCG5G8 (sterol

export proteins) in the enterocytes and hepatocytes (at the hepatobiliary interface). There are published studies that in people who have statin-induced over absorption of cholesterol and noncholesterol sterols, cholesterol in the artery wall is in part replaced by the more oxidizable and hence more atherogenic sterols. The authors of the STELLAR trial suggest that clinicians check these sterol markers on all patients as we could then pick better designed lipid-modulating therapies. Note that ezetimibe is the only FDA-approved product to reduce noncholesterol sterols.

Note: The views expressed in this article are those of the author. The National Lipid Association invites members to provide scientific and medical opinion that does not necessarily represent the policy of the NLA or its chapters.

[Attention readers: If you enjoy the case files of Dr. Dayspring, you can request a free subscription to his biweekly *Lipidaholics Anonymous Cases* newsletter by e-mailing the author at tdayspring@aol.com. Former cases are archived at www.lipidcenter.com under the “professionals” tab. New cases are now also posted on the NLA Community page at www.lipid.org under the “Lipidaholics” group. We encourage you to discuss this case and others with Dr. Dayspring at www.lipid.org. – ed.]

News & Notes: From the NLA Office

Reminder: 2010 Dues Are Due

We are happy to report that members are turning in their 2010 dues in strong numbers, expressing confidence in our Association. What's more, many are taking this opportunity to contribute to the Foundation of the NLA. To those who have paid their dues already, thank you from the NLA. To those who also contribute to the Foundation, be assured your donation will help us accomplish strong outreach to the public and further our association's mission. The NLA focuses on member education and the promotion of clinical lipidology as a medical specialty. The Foundation of the NLA directs its effort toward education and research that emphasizes professional, community and public health initiatives. Contributions to the Foundation are tax-deductible.

If you haven't renewed your dues yet, hurry so that you continue to receive the *Journal of Clinical Lipidology* and the *Lipid Spin*, and maintain your access to the NLA Community at www.lipid.org. Do renew your dues and ensure that you retain full member access to www.lipid.org, where you can always renew your membership directly.

W. Virgil Brown, MD, Now President of IAS

Having been President-Elect of the International Atherosclerosis Society (IAS), this year, past NLA President and current Editor-in-Chief of the *Journal of Clinical Lipidology*, W. Virgil Brown, MD, has begun his 3-year term as IAS President. The IAS is a coordinating body for some

62 national and regional organizations that are actively working on prevention of vascular disease throughout the world. It has organized a large triennial meeting of



scientists and thought leaders from the member organizations that brings together the latest in research-based knowledge about the causes and strategies for prevention of arteriosclerotic diseases. Research in clinical lipidology is a core component of these efforts. The NLA and the American Heart Association are the two American member organizations.

This is a natural position for Dr. Brown, who is actively working with our association's sister organizations around the world. From this new role as IAS President, Dr. Brown will be in an excellent position to further help the NLA form partnerships with lipid-focused specialty societies in Europe, Asia, South America and Australia.

Dr. Brown was able to work with Drs. Ernst Schaefer and Antonio Gotto in coordinating the International Symposium on Atherosclerosis (ISA 2009) in Boston last year, and he is already at work on ISA 2012 to be held in Sydney, Australia. As IAS President, Dr. Brown is now establishing a fund raising and new program committee to address new initiatives so that the organization can grow and better accomplish its mission of promoting international communication and promoting and dispersing knowledge regarding the study and treatment of

atherosclerotic diseases. We congratulate Dr. Brown and look forward to supporting him in his new position.

ACC Leads Push for CMS Review

The American College of Cardiology is taking an active lead to question the Centers for Medicare & Medicaid Services (CMS) planned cuts to Medicare payments to physicians for certain types of cardiovascular services. At issue is the data CMS collected to analyze its formula for physician expense reimbursement. The ACC pointed out some disparities in the methodology used in the decision to set the relative value for the services. ACC staff has produced compelling data and is asking CMS to consider this additional prior to enacting the reimbursement cuts.

The NLA is supportive of the ACC position and believes the impact of the cuts will reduce access to prevention services, the lipid clinic and patient care. It is our position that all valid practice data be considered before any cuts are implemented. In parallel with the effort to address CMS, legislation in the US House of Representatives (HR 4371) sponsored by Rep. Charles Gonzales [TX-20] has been drafted to alleviate some of the concerns regarding data collection.

More information about the cuts and their impact can be found at the ACC developed website:

www.campaignforpatientaccess.org/Legal. If you would like to comment on the impact of the cuts or the legislation,

News & Notes (continued on page 31)

Meeting Update: 2010 Scientific Sessions

The Latest Update on Plans for the 2010 Scientific Sessions

This year, the annual NLA Scientific Sessions will be held in Chicago, May 13–16, 2010. The Chairs of the 2010 meeting, **Drs. Peter Toth, Vera Bittner, Michael Davidson and Carl Orringer**, have planned an outstanding program. On Thursday afternoon, the Sessions will open with Keynote speaker, **Ira Tabas, MD, PhD**, whose address will explore “The Macrophage and Atherosclerosis.” Dr. Tabas is Professor of Medicine and Anatomy and Cell Biology (in Physiology and Cellular Biophysics) at Columbia University College of Physicians and Surgeons. The recipient of multiple awards from Columbia University and the American Heart Association, Dr. Tabas has lectured worldwide on the role of macrophages in atherosclerosis, and he has published numerous articles on cholesterol metabolism.

Those attending the Sessions are in for some surprises. This meeting will feature more panel discussions and special events than we’ve ever held before. Friday morning opens with the 3rd NLA Summit on HDL Therapeutics hosted by co-chairs **Peter Toth, MD, PhD**, and **Michael Davidson, MD**. Later in the day the focus will be on atherosclerosis, and **Drs. Steven Feinstein and Anthony DeFranco** will hold a debate on imaging, a major subject of discussion in our field. As another first for the Sessions, on Friday evening we’re holding a fellows mentoring session and reception, underscoring the collegiality and professional development opportunities

that distinguish our association.

Saturday starts early at breakfast, where NLA thought leaders and Sessions faculty will be on hand for “Meet the Experts” roundtables. Come prepared with your questions. The morning Sessions will cover nutrition and diet and then will turn to critical issues in clinical lipidology, such as the usefulness of non-HDL-C and ApoB in risk assessment, LDL particle size and contrasting opinions on current lipid lowering therapies featuring a debate with **Drs. W. Virgil Brown and Steven Nissen**. Arrive early for this one.

After lunch, attend the poster sessions and select one of the focused breakout presentations to attend. These are always popular and allow you to tailor the program to meet your interests, in this case choosing between clinical trial evaluation, methods for achieving TLC compliance, and management of statin-intolerant patients. Later in the afternoon, we’re holding a special Session on Familial Hypercholesterolemia (FH), and the importance of cascade screening and emerging therapies, co-sponsored by MEDPED (Making Early Diagnosis, Preventing Early Deaths) and the Foundation of the NLA. In the evening join the pomp and circumstance of the ABCL/ACCL Convocation for new Diplomates, and attend the NLA Foundation Benefit Gala Event.

Stay through Sunday so you can attend the interactive case presentations (prepare now by seeing virtual patients at www.lipid.org/vclinic). Special insights will be offered by **Alan Brown, MD, Carl**



Register online and discuss the Scientific Sessions at www.lipid.org

Go to “Topics” and look for “2010 Scientific Sessions.”

Orringer, MD and Anne C. Goldberg, MD. The meeting will conclude with a 2-hour condensed version of the Lipid Clinic Operations Course conducted by **Ralph LaForge, MSc**. As you can see, this program will feature a combination of high-science and clinical presentations, ensuring solid offerings for all attendees. More details will be posted at www.lipid.org/sessions as they become available. See you in Chicago!

Call for Abstracts: New Deadline

If you’re planning to submit a poster for this year’s NLA Scientific Sessions, make a note that we will need your abstract soon. We have extended the deadline for abstract submissions to **March 15, 2010**. Accepted abstracts will be published in the May/June 2010 issue of the *Journal of Clinical Lipidology*.

Presenters who are Young Investigators (with accepted abstracts) will receive a \$300 travel voucher and free registration to the 2010 NLA Scientific Sessions. Submissions will also be eligible for the NLA Young Investigator Award. For more details on topics and to submit your abstract online, visit www.lipid.org/abstracts.

NATIONAL LIPID ASSOCIATION 2010 ANNUAL SCIENTIFIC SESSIONS



May 13-16, 2010 • Sheraton Hotel & Towers

www.lipid.org

Provided in conjunction with the Midwest Regional Chapter

Call for Abstracts—DEADLINE EXTENDED

The new deadline for posters to be submitted is **MARCH 15, 2010**. All accepted poster abstracts will be published in the May/June 2010 issue of the *Journal of Clinical Lipidology*. For guidelines and to submit an abstract visit www.lipid.org/poster

Featured Ancillary Courses

- Lipid Management Training Course – Updated Curriculum for 2010!
- Cardiometabolic Risk Reduction Through Weight Management – New Curriculum for 2010!
- Masters in Lipidology™ Course – New Curriculum and Format for 2010!

Program Highlights

Back By Popular Demand

- 3rd NLA Summit on HDL Therapeutics
- Point/Counterpoint Debates
- Challenging Case Presentations
- Poster Sessions & Young Investigator Award
- Practical Breakout Sessions

New This Year

- Exhibitor Theater and Innovation Labs
- NLA Foundation Heart Healthy Events
- Meet the Faculty Roundtable Session
- Trainee Mentoring

2010 Program Co-Chairs

Peter Toth, MD, PhD

President
Midwest Lipid Association

Vera Bittner, MD, MSPH

President
National Lipid Association

Michael Davidson, MD

President-Elect
National Lipid Association

Carl Orringer, MD

President-Elect
Midwest Lipid Association



Register now at
www.lipid.org/sessions



NLA 2010 ANNUAL SCIENTIFIC SESSIONS
PROVIDED IN CONJUNCTION WITH THE MIDWEST REGIONAL CHAPTER
MAY 13-16, 2010
SHERATON HOTEL AND TOWERS • CHICAGO, IL

Registration

1

 First Name Middle Initial Last Name

 Mailing Address

 City State or Province Zip Country

 Phone Emergency Contact/Phone

 Email

Check all that apply: MD DO PhD RN NP PA RPH PharmD RD Other _____

Guest name(s), if attending meeting:

Membership status:

- I am currently a member.
 My application for membership has been submitted and confirmed.
 I will apply at www.lipid.org
 Please send me membership information.

2

| Circle fee based on attendee type | NLA Member Discount Rate | Registrant Rate | Trainee Rate | Industry Rate |
|---|--------------------------|-----------------|--------------|---------------|
| <input type="checkbox"/> Annual Scientific Sessions May 13-16, 2010 Includes course syllabus and one admission badge to Exhibit Hall for all food functions. <i>(Saturday Night Dinner NOT Included)</i> | \$495 | \$795 | \$75 | \$795 |
| <input type="checkbox"/> Join NLA and register for Annual Scientific Sessions | N/A | \$595 | \$75 | N/A |
| Ancillary Courses | | | | |
| <input type="checkbox"/> Master's Board Review Course* May 12-13, 2010 | \$695 | \$895 | \$595 | \$1,200 |
| <input type="checkbox"/> Lipid Management Training Course May 12-13, 2010 | \$395 | \$585 | \$195 | \$900 |
| <input type="checkbox"/> Comprehensive Cardiometabolic Risk Reduction Program May 13, 2010 | \$0 | \$0 | \$0 | \$0 |
| Registration Fee Total | \$ _____ | \$ _____ | \$ _____ | \$ _____ |

3

| | | | | |
|---|-------|----------|----------|--|
| Social Events and Guest Fees | | | | |
| <input type="checkbox"/> Saturday Night Dinner-Registrant | \$125 | | \$ _____ | |
| <input type="checkbox"/> Saturday Night Dinner- Guest(s) | \$125 | X _____ | \$ _____ | |
| <input type="checkbox"/> Exhibit Hall Pass-Guest(s) | \$80 | X _____ | \$ _____ | |
| Social Events and Guest Total | | \$ _____ | \$ _____ | |
| Combined Total Sections 2,3 & 4 | | \$ _____ | \$ _____ | |

5

PAYMENT METHOD

- VISA MC
 AMEX Check

Make checks payable to the NLA

 Credit Card # Exp. Date

 Signature

 Name on Card

3 EASY WAYS TO REGISTER



Mail To:

National Lipid Association
 6816 Southpoint Parkway, Suite 1000
 Jacksonville, FL 32216



Fax to:

NLA at 904-998-0855
 Fax with credit card number and signature



Online:

www.lipid.org

IMPORTANT INFORMATION

***Master's Course**

To purchase related study materials or the NLA-SAP's please go to:
www.lipid.org/education/nlasap.

Registration: Registration and payment must be received no later than April 29, 2010. After this date a syllabus and name badge cannot be guaranteed- so register TODAY!

Cancellation: Telephone Cancellations will not be accepted. A written notice of cancellation must be received no later than April 29, 2010. This includes Social Events and Guest Fees.

Special Needs:

ADA Compliance:

Attendees who need additional reasonable accommodations or who have special needs should contact the NLA at 904-998-0854.

Foundation Update: Recognizing our donors



FOUNDATION
of the National Lipid Association

The membership of the NLA is a uniquely collegial and supportive group of professionals who know the value of teamwork. We asked for donations from members to help launch our Foundation and to date we have received \$8700. Our thanks go to the following contributors, and we hope you will take an opportunity to join them in supporting your association's Foundation:

Founding Donors

- Anne C. Goldberg, MD
- Carl Orringer, MD

Contributing Donors

- David Akin, MD
- Petar Alaupovic, PhD
- Virginia Bailey, RN
- Paul Bekx, MD
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- Jose Vazquez-Tanus, MD
- Luis Villamon, MD
- Steven von Elten, MD
- Thomas R. White, MD
- Gordon Wolfe, MD
- Thomas Zoch, MD

Especially in today's difficult economy, your support means so much. All donors to the Foundation of the National Lipid Association receive our thanks.

Foundation Events at NLA Meetings

You also have the opportunity to help the Foundation by attending special events at our Clinical Lipid Update meetings and the annual Scientific Sessions. For example, the Vines & Vessels Gala Event at our Winter meeting in San Francisco. This makes it easy to do your part and have a ball at the same time. Specific details will be listed on our programs and registration forms, both in print and online.

Foundation Grant Program

The Foundation's grant program provides education, research and community outreach grants to applicants who meet the qualifying criteria. The Foundation will fund grants up to \$5000 or less. Applications must be submitted at least 60 days prior to the date funds are needed, and funds must be used for activities within 90 days of grant approval. To apply online or learn more, log onto www.lipidfoundation.org and click on "Grants." If you have any questions, please call **Karen Kent** at 904.309.6211

Foundation Update (continued on page 30)

NLA Community: Introduction to the “Topics” section

NLA Community Update: What Have You Done for Us Lately?

Past articles in this series have focused on the exciting new features and content available to members at the online home of the National Lipid Association at www.lipid.org. This time, instead of focusing on what we can do for you, we’re going to show you how to contribute to lipidology’s workspace on the web.

The new NLA Community website at www.lipid.org features multiple opportunities for you to not just be a practitioner of our discipline; but to be a determiner of its future. This article introduces you to the “Topics” section.

What Are “Topics”?

Topics, on the NLA website, resemble that familiar Internet tool, the Discussion Forum. Also called Bulletin Boards, discussion forums are nothing new to the Internet; and it is precisely this familiarity that makes “Topics” a great place to start participating.

To begin with, you’ll need to be logged in. If you don’t have your login credentials, or need help getting signed in, send an e-mail to webmaster@lipid.org or give **Clark Morgan** a call at 904-309-6202.

As of this issue, the clinical content published in the Lipid Spin will be linked to established discussions in the Topics area of the website. After reading an article in the Lipid Spin, look for the “NLA Community” logo directing you to the website Topics area and browse for the article title in the master list. If you don’t

see the article you’re looking for, you can hit [Ctrl]+[F] from any web browser to search for it on the current page (note that the discussion you are looking for might be pushed to a second page by current activity).

Easy Tip: Read the articles online and then link directly to the discussion.

The NLA encourages authors to participate in these discussions. Joining in discussion forums is a valuable way for members to query authors directly about their work. In many cases, our authors will have been the lead investigators on the studies presented. Ask, answer and debate with your colleagues in an interactive and professional environment.

Easy Tip: Take advantage of the “customize my home page” feature discussed in our last issue to see quick links to updated forum discussions every time you login to lipid.org. Or visit the Quick Tips section of the home page at lipid.org.

Take the Lead

The Topics area is not limited to discussions of current Lipid Spin articles. Do you have a question of your own that is not covered by the topics currently available? Feel free to jump in and start a new topic. Just click “New Topic” from the main Topics page and post your question or observation. If you know someone who might be interested, browse the members area of the site and invite them to join for comment.

The National Lipid Association is working hard to make the new NLA Community

a resource all our members can use and enjoy. But the real value comes from you, the NLA member. Visit us on the web often at www.lipid.org. ■

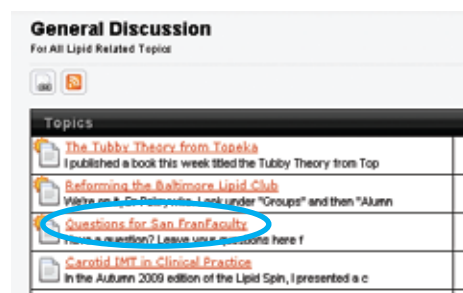
1) After selecting “Login” at top right, enter your username and password.



2) Select “Topics.”



3) Select a topic to read from the list. Newest topics will be at the top.



4) After reading the comments, you can add one yourself by selecting “Post Reply.”



We know you strive for excellence in the field.



Why not demonstrate your expertise by seeking certification in Clinical Lipidology?

The ACCL* Offers Two Pathways to Recognition:

- The **Clinical Lipid Specialist (CLS)** program is an advanced **certification** pathway open to licensed Allied Health Professionals specializing in lipid management.
- The **Basic Competency in Clinical Lipidology (BCCL)** program offers a **competency assessment and credentialing** pathway for any healthcare professional or paraprofessional with an interest or involvement in the area of dyslipidemia.

2010 CLS EXAMINATIONS:

NEW IN 2010 - Exam to be offered in computerized testing centers across US and Canada

Spring Testing Window
March 15–April 15, 2010
(Application Deadline:
February 26, 2010)

Summer Testing Window
June 14–July 14, 2010
(Application Deadline:
May 17, 2010)

Fall Testing Window
September 20–October 20, 2010
(Application Deadline:
August 30, 2010)

2010 BCCL EXAMINATIONS:

Exam is offered in computerized testing centers across US and Canada year-round

Each pathway offers a unique application process and examination that will assess and validate the specialized knowledge and training required to practice or work in the multifaceted and unique field of lipidology. Both exams are offered electronically at testing center locations around the US and Canada for maximum convenience and cost-effectiveness.

Certification and credentialing by the ACCL demonstrates your professional commitment to the prevention of cardiovascular disease and documents your expertise in lipid management to patients, referring professionals, employers and colleagues.



The Accreditation Council for Clinical Lipidology

For more information visit:
www.lipidspecialist.org

*The **Accreditation Council for Clinical Lipidology (ACCL)** is an independent certifying organization that has developed standards and an examination in the field of Clinical Lipidology for the growing number of **allied health professionals** who manage and treat patients with lipid and other related disorders.

Phone: 904-309-6250

Meetings & Courses Calendar



Online Activities

Available at www.lipid.org/education

Lipid Insights Virtual Journal Club

Should an LDL-C Less Than 70 mg/dL and a CRP Less Than 2 mg/L Be Dual Targets of Therapy? Pro and Con
Sponsored by the NLA

Lipid Insights from AHA 2009: Implications for Clinical Practice
Sponsored by the NLA

Implications of the ARBITER-6 HALTS Trial
Sponsored by the NLA

NLA Virtual Lipid Clinic

(www.lipid.org/vclinic)

3 Patients Waiting

Online Self-Assessment Programs

Four Complex Lipid Management SAPs are available at www.lipid.org/education:

- Clinical Applications of Advanced Lipoprotein testing, Inflammatory Markers and Non-invasive Assessment of Atherosclerosis
- Management of Low HDL-C
- Management of Dyslipidemia—Focus on Combination Therapies
- Pharmacology and Safety of Lipid-Altering Therapies

2010 Meetings & Courses

March 14–16, 2010
American College of Cardiology
59th Annual Scientific Sessions
Atlanta, GA
www.acc.org

April 15–17, 2010
Preventive Cardiovascular Nurses Association
16th Annual Symposium
Northwest Chicago, IL
www.pcna.net

April 21–25, 2010
American Association of Clinical Endocrinologists
19th Annual Meeting and Clinical Congress
Boston, MA
www.aace.com

May 13–16, 2010
NLA 2010 Annual Scientific Sessions
Hosted by the Midwest Regional Chapter
Sheraton Hotel and Towers - Chicago, IL
www.lipid.org

June 25–29, 2010
American Diabetes Association
70th Scientific Sessions
Orlando, Florida
professional.diabetes.org/

August 27–29, 2010
NLA Summer Clinical Lipid Update
Hosted by the Southeast & Northeast Regional Chapters
Mayflower Hotel - Washington, DC
www.lipid.org

November 13–17
American Heart Association Scientific Sessions 2010
Chicago, IL
scientificsessions.americanheart.org

NLA Professional Development Courses

May 12–13, 2010

- Lipid Management Training Course
- Comprehensive Cardiometabolic Risk Reduction Course - Phase III
- Masters in Lipidology Course
Sheraton Hotel and Towers - Chicago, IL
www.lipid.org/education

August 26–27, 2010

- Lipid Management Training Course
- Comprehensive Cardiometabolic Risk Reduction Course - Phase III
- Masters in Lipidology Course
Mayflower Hotel - Washington, DC
www.lipid.org/education



Come to Washington D.C. for the Summer Clinical Lipid Update

Clinical Feature (page 7)

raises HDL levels by as much as 30 to 35 percent. The use of nicotinic acid is often limited by poor tolerability. Side effects can include flushing, pruritus, paresthesias, nausea, and liver toxicity. It also has some adverse metabolic effects, such as hyperglycemia, hyperuricemia, hypotension in patients on vasodilators, and elevation in plasma homocysteine levels.

Omega-3 fatty acids (fish oil) is an effective agent to reduce plasma triglyceride by decreasing hepatic VLDL synthesis and increasing LPL activity. Daily consumption of 4 g of omega-3 fatty acids, along with with restricted energy and saturated-fat intakes, can reduce plasma triglyceride levels by 19-44%.²⁰ Omega-3 fatty acids constitute only 30 to 50 percent of many fish oil supplements, however. In comparison, the commercial preparation (Lovaza in the United States and Omacor elsewhere) is 85 percent omega-3 fatty

acids. Each capsule of Lovaza (1g) contains about 465 mg EPA and 375 mg DHA, so 4 capsules daily is the recommend dose. The US FDA limited approval of Lovaza to the treatment of severe hypertriglyceridemia (≥ 500 mg/dL) because of concerns that it appears to increase LDL-C levels.

Orlistat is a lipase inhibitor developed for the treatment of obesity and associated comorbidities. Its inhibition of fat absorption can lower plasma triglyceride levels, which was demonstrated in several clinical trials.²¹⁻²³ Orlistat can be used alone or in combination with fibrates in patients with HTG. However, patients need to be on a strict low-fat diet, and poor tolerance may limit its clinical use.

Summary

Severe HTG (especially TG > 1000 mg/dL) is a significant risk factor for acute pancreatitis. It generally requires acute management with the patient kept NPO and given IV insulin and other treatments

to lower TG level quickly. These patients usually need long-term oral agents such as fibrates, niacin and omega-3 fatty acids to maintain plasma TG below 500 mg/dL. Moderate HTG maybe a risk factor for cardiovascular diseases. However, the evidence of benefits to using TG-lowering agents such as fibrates is weak. The NCEP-ATP III recommendations for the treatment of hypertriglyceridemia are:

- (1) TG 150–199 mg/dL: lifestyle modification;
- (2) TG 200–499 mg/dL: non-HDL cholesterol becomes a secondary target of therapy after LDL cholesterol. Non-HDL < 130 mg/dL for CHD or equivalent; < 160 for ≥ 2 CHD risk factors and < 190 for 0–1 CHD risk factors; and
- (3) if TG > 500 mg/dL, the primary goal is to prevent pancreatitis by medical therapy.⁸ ■

Practical Pearls (page 9)

fat intake that, in predisposed pregnant women, usually begin or worsen in the latter part of the first trimester of gestation. Those at risk usually have varying degrees of lipoprotein lipase deficiency, a personal and often a family history of strikingly elevated triglyceride levels after an overnight caloric fast. In these conditions, plasma triglyceride levels typically rise well above 700–1000 mg/dL or greater after intake of a fatty meal.

What changes in triglycerides occur normally in pregnancy?

Plasma triglyceride levels normally increase progressively over the course of gestation. These elevations in plasma triglycerides stem both from an estrogen induced stimulation of hepatic release of triglyceride-rich lipoproteins and to an inhibition in plasma lipoprotein lipase activity that slows the clearance of plasma triglycerides. During the first trimester of pregnancy, increased ovarian estrogen production is mainly responsible for the progressive elevations in plasma

triglycerides. The subsequent secretion of placental estrogen and potentially other placental hormones likely provide the additional modest augmentation of triglyceride levels that are present over the next 2 trimesters. The prolonged presence of strikingly high concentrations of chylomicrons greatly intensifies the risk for a life-threatening bout of acute pancreatitis.

Episodes of acute pancreatitis pose considerable risk of fetal demise and also threaten the life of the mother. ■

Member Spotlight (page 12)

Dr. deGoma cautions. “You may have been speaking with a patient for 5 or 15 minutes on an issue. I use customized forms to shorten the time needed for writing and documentation.” He also has developed an impressive website at www.deGomaMD.com, where you can view many of his patient counseling materials.

He asks his patients directly, “You tell me how much prevention you want—40%? You are at high risk, almost the same as if you were in secondary prevention. Do you know someone who died suddenly of heart attack? We have to lose some weight, change your profile, get exercise, stop smoking.” Dr. deGoma adds, “I can show how they can reduce their risk by 70%—and nobody ever says, ‘I only want 0% reduction. Given the choice, most high risk patients will choose the maximum possible evidence-based prevention.’” The ultimate goal of treatment, after all, is to reduce events.

“Heal thyself”

What happens to a practice with many high-risk patients after 9 years of aggressive lipid therapy? “It’s incredible!” Dr. deGoma says. In 2004, Dr. Michael Wolk, then president of the ACC, published an editorial titled, *The Promise of Prevention—So, why aren’t all cardiologists preventive?* This was an encouragement to Dr. deGoma, as it underscored his being on the right track, evolving his practice as cardiology itself continues to evolve.

Another important way he motivates his patients is by sharing with them his own cardiology experiences. Taking advantage of an opportunity to take a free EBT test for coronary artery calcification, he found that he had a significant calcium score, and so Dr. deGoma became his own patient. “I have experienced muscle aches and muscle weakness with 2 of the 3 potent statin drugs available. I discontinued

niacin 3 times due to almost unbearable itching and flushing but persevered on the fourth attempt (after the EBT) and overcame the side effects after 6 months. I used aggressive combination therapy to halt disease progression and hopefully, even cause some regression by reducing my LDL-P below 1,000, my LDL-C below 70 and raising my HDL-C to over 50,” he says. This helps him both empathize with the challenges his patients face, and gives him credibility with them. He tells a patient that, “I can do for you what I do for myself,” he becomes both a reliable source of information to the patient and also a behavioral motivator.

The future of CVD prevention

Clinical trials of statin medications often show a 40% reduction in risk. “The 4S trial ended after 5 years, the JUPITER trial ended after less than 2 years, but in practice you don’t stop,” Dr. deGoma says. “So in clinical practice, you see the clinical events dropping steadily.” He founded NJ Preventive Cardiology and Cholesterol Clinic in 2004, with the thought of incorporating lipid treatment in a regular cardiology practice. “The traditional lipid clinic model cannot treat the over 40 million Americans eligible for lipid drug treatment, even more if you add in those at intermediate risk with significant coronary calcification, high hs-CRP or increased CIMT. We should empower all medical practices to become basic lipid clinics. The solution is more education and a widely available, cost-effective preventive care delivery system that minimizes regional variations in quality.” Speaking of which, Dr deGoma became a board member of the Northeast Lipid Association, and he has worked to help the NLA be a strong voice for CVD prevention. A Diplomate of the American Board of Clinical Cardiology, he is also a Fellow of the NLA.

Dr. deGoma knows he is ahead of many in his profession, but notes that according

to the Institute of Medicine, it takes an average of 16 years for a new mode of treatment to become widely implemented in clinical practice. “We need changes in reimbursement, to create more incentives for physicians, patients and insurance companies,” he says, “Some of my colleagues thought I was crazy when I started. Cholesterol wasn’t part of my training. I was too busy in my practice to acquire new skills. The young generation of physicians have a chance to get on board this concept.”

In cardiology, the goal of treatment is relief of symptoms, such as shortness of breath in congestive heart failure. Dr. deGoma observes that, “Prevention is a different approach. It requires reaching specific numeric targets, not symptom relief, and we’re not used to that. After 5 years, I’ve developed a numerically goal-oriented clinical information management system.” The future of cardiology, he feels, will not lie in more aggressive intervention, but in more aggressive prevention. He summarizes his experience by saying, “My personal and professional journey to prevention took longer than my 3-year cardiology fellowship more than 25 years ago, but it changed the way I practice cardiology. Prevention is the missing component of cardiac care.” ■

For clinical tips and downloads, please visit www.deGomaMD.com/PracticalTips.htm. Dr. deGoma discusses the following topics:

1. How to use the Cyber Dietitian.
2. How to make yourself known in your community as a certified lipidologist—putting all the pieces together.
3. EZ Daily LDL Chart Audit
4. EZ Cardiovascular Risk Assessment

NLA President (page 1)

may enhance your own practice.

In addition, the new NLA Community website is up and running. We're still making small adjustments here and there and you'll occasionally notice changes, but this is now a full-fledged interactive forum where you can gain a tremendous amount of information about the NLA and also discuss issues with your colleagues, ask and answer questions, search our library

of slides and much more. We will also continue to hold walk-through sessions for members so watch your e-mail for the next session available. In the meantime, feel free to explore and investigate on your own, and each issue of the *Lipid Spin*, including this one, has a "how-to" article describing a feature of the site to encourage your use of it.

Lastly, consider some strategic planning for yourself. If you aren't certified by

the ABCL or ACCL, make this a year to take steps toward credentialing. Many of your colleagues have passed the exam so prepare to join them. Visit the NLA website at www.lipid.org/education to learn more. The NLA is helping members to make the right connections but ultimately it's up to you to bring knowledge into your practice and community. ■

Cardiology Corner (page 10)

When atherosclerosis is present, it may be non-obstructive on angiography. Only 40% of the women in the WISE Study who underwent angiography for evaluation of chest pain or an abnormal stress test had a flow-limiting lesion. Pathologic studies and intravascular ultrasound reveal that outward remodeling is more common in women than men, resulting in angiographic underestimation of atherosclerotic burden. The traditional disease management

approaches that focus on critical stenosis often fail to identify those women at critical risk for myocardial ischemia. The initial presentation of ischemic heart disease in 60% of women will be an acute myocardial infarction or sudden death. At autopsy, women are more likely to have plaque erosion rather than plaque rupture as the cause of the acute event. The five-year annualized event rates were 16% in WISE women with non-obstructive CAD and 7.9% in WISE women with normal

coronary arteriography.

Symptomatic women, more than men, have persistent symptoms requiring repeat hospitalizations, repeat diagnostic evaluation and lower ratings of general well being. Treatment should be aimed at traditional risk factors to reduce atherosclerotic burden and improve endothelial dysfunction. Continued research is needed to improve symptoms and reduce cardiovascular risk. ■

Lipid Luminations (page 14)

cholesterol and triglycerides, and are exacerbated by excessive body mass index. It is possible that aggressive treatment of these risk factors, beginning in middle age, might delay or prevent the appearance of CAD. It has been shown that men with

optimal risk factor levels at age 50 have a lifetime risk of CAD of 5% vs. 69% in those with 2 or more risk factors. In women, the corresponding risks are 8% and 50%.⁵

Lipid management is critical if we are to prevent the progression of subclinical coronary atherosclerosis to symptomatic

CAD. Tools such as CAC scoring can be utilized to identify patients for whom optimal lipid management is indicated. Many patients with low or intermediate risk using standard risk assessment tools will be found to have higher risk based on their CAC score. ■

Foundation Update (page 24)

Community Outreach

As a special project for 2010, the Foundation has formed a writing committee to produce a book for the general public and patients about cholesterol. When published, we'll be able to offer it to NLA members at discount, and this will be an excellent resource

for practitioners and their patients for advanced counseling. The members donating their time to this project have our sincere appreciation.

Support the FNLA

Helping to fund the Foundation of the National Lipid Association grant program is rewarding, because it's an opportunity to give something back to your profession

while also promoting the field of lipidology. Take a moment now to invest in the Foundation. You can make a tax-deductible donation online at www.lipidfoundation.org, contribute when you renew your dues, call us at 904.309.6260, or send your check made out to "Foundation of the NLA" to National Lipid Association, 6816 Southpoint Pkwy, Ste 1000, Jacksonville, FL 32216. ■

News & Notes (page 20)

the NLA has set up a discussion area set up in the “Topics” portion of our community website at www.lipid.org.

FROM THE EDUCATION DEPARTMENT

New CLM SAPs (CME/CE)

It's easier than ever to participate in our Complex Lipid Management Self-Assessment Programs (CLM SAPs) and self-study modules (NLA-SSMs)—four of them are now online activities for your convenience. You can quickly gain 5~6 CME/CE and further sharpen your skills and assess areas for improvement. The online modules are available at www.lipid.org/education.

By now you've received our newest CLM-SAP in the Series, Edition 13—*Challenging Cases in Dyslipidemia*, which was mailed together with the Nov/Dec 2009 edition of the *Journal of Clinical Lipidology*. This will also be available as an online activity by spring 2010.

See Your Patients at the Virtual Lipid Clinic (CME/CE)

The NLA vClinic™ is the future of online CME. The NLA vClinic at lipid.org lets you diagnose and manage virtual patients over multiple visits with input from leading clinical experts, all while earning CME/CE credit. Be sure to use the vClinic CMECompanion that accompanies these activities to assess your clinical mastery and compare your performance with other learners. Visit www.lipid.org/vclinic to participate. We are adding new patients frequently so come back if you haven't visited lately. We now discuss these cases at our Clinical Update Meetings, so

investigate the vClinic and be prepared in advance.

Fellows Training and Mentoring

The NLA Graduate Medical Education Committee is developing a basic clinical lipidology Fellowship Curriculum. This is a necessary step in order to apply for approval by the ACGME. This in turn takes the NLA one step closer to establishing clinical lipidology as a recognized specialty of medicine by the American Board of Medical Specialties.

We have completed a web-based in-service exam on lipid metabolism and dyslipidemia management to teach training guidelines for fellowship programs and comply with the ACGME mandate for outcomes assessment. The exam is derived from the NLA Self-Assessment Program, featuring 50 multiple-choice questions adapted from the NLA-SAP, Volume I. Program Directors can track their Fellows' performance on the exam and compare their program's overall performance with that of their peers. The NLA-SAP, Fellows Edition is now available to all Program Directors through the American College of Cardiology Training and Workforce Committee. Any members involved in Fellows training may contact the NLA office at fellows@lipid.org to complete an application to access this program.

Fellows Outreach

Our 2010 Fellows Outreach Program is being held in conjunction with the three scientific meetings scheduled for 2010. The NLA will invite Program Directors in the hosting regions to send their Fellows to participate in the NLA Lipid Management Training Course, and also attend a mentoring session and reception with faculty and Program Directors.

There is no fee for attending, and travel scholarship grants (up to \$500) will be provided to help cover the cost of Fellow travel and accommodations. Contact the NLA office at fellows@lipid.org for more details about this program.

Give Us Your Opinion: Annual NLA CME Needs Assessment Survey

Soon we will be sending you an invitation to participate in our annual CME Needs Assessment Survey. This is your opportunity to tell us what you need most in education, and how we can best meet your needs. We directly apply your preferences in the design of our educational activities—as shown by our burgeoning range of Lipid Insights Webcasts, the NLA vClinic, and online study and self-assessment modules, all of which you asked for. Do take a few minutes when you see the survey invitation and help us to help you. Incentives will be offered to those who participate.

Comprehensive Cardiometabolic Risk Reduction Course—Phase III

The latest version of this popular program focuses on the practical aspects of office-based weight management for patients with cardiometabolic risk. Weight loss approaches in cardiometabolic risk reduction are covered extensively, with expert insights and guidance on best outcomes. The 3-hour course includes case studies improvement strategies to help you manage standards-based performance goals for various patient populations. The course will be offered three times in 2010: February 19 in San Francisco, CA; May 14 in Chicago, IL; and August 27 in Washington, DC. For more information and to register, visit www.lipid.org/education. ■

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