AHA Redefines Triglyceride Target as 100 mg/dL

BY JENNIE SMITH

FROM CIRCULATION

Triglyceride levels, which play a large role in both atherosclerotic risk and metabolic health, are highly responsive to decreases in dietary sugar intake and saturated and trans fat intake, along with increases in omega-3 acid intake and exercise, according to a scientific statement from the American Heart Association.

“What’s new is that we point out that triglycerides might be considered a marker for metabolic health,” Dr. Neil J. Stone of Northwestern University Chicago, vice-chair of the statement’s writing group, said in an interview. “If you have a country where you’re seeing more obesity and more diabetes, it becomes important for people to start asking themselves ‘are there signs that I should be doing something different?’ and this is one,” he said.

The scientific advisory, citing some 528 sources, was not presented as a clinical guideline so much as a distillation of 30 years worth of evidence on the complex relationship among lifestyle factors, triglycerides, and cardiovascular and metabolic health (Circulation 2011; doi:10.1161/CIR.0b013e3182160726).

However, the authors, led by Dr. Michael Miller, director of the Center for Preventive Cardiology at the University of Maryland, Baltimore, included a number of recommendations on diagnosing and treating hypertriglyceridemia, focusing on dietary and lifestyle changes.

The statement emphasizes the “increasingly crucial role” of triglycerides in the evaluation and management of cardiovascular disease, and the importance of diet—including consumption of sugars common in beverages—in contributing to unhealthy triglyceride levels.

Reductions of 50% or more are achievable without the use of medication—a finding that is not widely accepted by some clinicians and triglycerides are still lacking crucial clinical trial evidence,” Dr. Miller and colleagues wrote in their analysis, noting that certain medications, including hormonal treatments, can also contribute to elevated triglycerides.

About a third of American adults have elevated triglyceride levels, which are defined as fasting triglyceride of 150 mg/dL or higher. The authors recommended that optimal fasting triglyceride levels now be defined as 100 mg/dL—and that clinicians screen initially for nonfasting triglyceride, defining normal at below 200 mg/dL. People with higher nonfasting levels may then be further screened for fasting triglyceride.

The new dietary recommendations include restricting added dietary sugar to 7%–10% percent of calories consumed. In support of this, the authors cited a study of 6,113 U.S. adults showing that the lowest triglyceride levels were observed when added sugar represented less than 10% of total energy, and that higher triglyceride levels corresponded with added sugar accounting for a greater proportion of energy intake (JAMA 2010;303:1490-7).

The authors singled out fructose as particularly problematic. Fructose in excess of 100 g/day, and possibly in excess of 50 g/day, has been associated with raised triglyceride levels. A typical can of cola or lemon-lime soda contains more than 20 g of fructose, the authors noted.

Dr. Miller and his colleagues advocated weight loss of 5%–10% of body weight, which is associated with a 20% reduction in triglycerides, and regular aerobic exercise, to reduce triglyceride levels closer to optimal. They also promoted increasing dietary fiber, keeping saturated fat below 7% of calories, eliminating trans fat from the diet, and increasing omega-3 polyunsaturated fatty acid consumption in the form of marine fish.

Funding for the statement was provided by the American Heart Association. Dr. Miller declared no conflicts affecting the drafting of the statement. Dr. Stone and the report’s third author, Dr. Christie Ballantyne of Baylor College of Medicine in Houston, disclosed support from pharmaceutical industry sources. Other coauthors and some reviewers disclosed additional support from pharmaceutical and agricultural firms.

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Indications and Usage
Norditropin® (somatropin [rDNA origin] injection) is indicated for the treatment of children with growth failure due to inadequate secretion of endogenous growth hormone, the treatment of children with short stature associated with Noonan syndrome or Turner syndrome, the treatment of children with short stature born small for gestational age (SGA) with no catch-up growth by age 2-4 years, and for the replacement of endogenous growth hormone in adults with growth hormone deficiency (GHD) who meet either of the following two criteria: 1. Adult Onset: Patients who have GHD, either alone or associated with multiple hormone deficiencies (hypopituitarism), as a result of pituitary disease, hypothalamic disease, surgery, radiation therapy, or trauma; or 2. Childhood Onset: Patients who were growth hormone deficient during childhood as a result of congenital, genetic, acquired, or idiopathic causes.

Important Safety Information
Somatropin should not be used to treat patients with acute critical illness due to complications following open heart or abdominal surgery, multiple accidental trauma or acute respiratory failure as increased mortality may occur. Somatropin is contraindicated in patients with Prader-Willi syndrome who are severely obese, have a history of upper airway obstruction or sleep apnea, or have severe respiratory impairment. There have been reports of sudden death when somatropin was used in such patients. Norditropin® is not indicated for the treatment of patients who have growth failure due to genetically confirmed Prader-Willi syndrome.

Somatropin should not be used or should be discontinued with any evidence of active malignancy. Any preexisting malignancy should be inactive and its treatment complete prior to instituting therapy with somatropin. Somatropin should be discontinued if there is evidence of recurrent activity. Patients with preexisting tumors or GHD secondary to an intracranial lesion should be monitored routinely for progression or recurrence. In childhood cancer survivors, an increased risk of a second neoplasm, particularly meningiomas in patients treated with radiation to the head for their first neoplasm, has been reported in patients treated with somatropin.

Somatropin should not be used in patients with active proliferative or severe non-proliferative diabetic retinopathy, for growth promotion in pediatric patients with closed epiphyses, or in patients with known hypersensitivity to somatropin or any of its excipients.

Somatropin may decrease insulin sensitivity particularly at higher doses in susceptible patients. Glucose levels should be monitored periodically, including close monitoring of patients with preexisting diabetes or glucose intolerance. Doses of anti-hyperglycemic drugs (insulin or oral agents) may require adjustment for patients with diabetes on somatropin therapy. Intracranial hypertension (IH) with papilledema, visual changes, headache, nausea and/or vomiting, usually occurring within the first eight (8) weeks after initiation of somatropin therapy, has been reported in a small number of patients. In all reported cases, rapid resolution has occurred after therapy cessation or a reduction of dose. Funduscopic examination should be performed routinely before and during somatropin therapy. If papilledema is observed, somatropin treatment should be discontinued.

Fluid retention during somatropin replacement therapy can occur in adults more frequently. Clinical manifestations of fluid retention are usually transient and dose dependent. In patients with GHD, central (secondary) hypothyroidism may first become evident or worsen during somatropin therapy. Periodic thyroid function tests are recommended and thyroid hormone replacement therapy should be initiated or adjusted as needed.

Slipped capital femoral epiphysis may occur more frequently in patients with endocrine disorders (including GHD and Turner syndrome) or with rapid growth. Onset of a limp or complaints of hip or knee pain in somatropin patients should be carefully evaluated. Rapid growth may also result in progression of preexisting scoliosis. Patients with a history of scoliosis or skeletal abnormalities, which may persist in untreated Noonan, Turner or Prader-Willi syndrome, should be monitored. Patients with Turner Syndrome should be evaluated carefully for otitis media and other ear disorders since these patients have an increased risk of ear and hearing disorders. Somatropin treatment may increase the occurrence of otitis media in patients with Turner syndrome. Somatropin may also increase the risk of IH in Turner patients. In addition, patients with Turner syndrome should be monitored closely for cardiovascular disorders (e.g., stroke, aortic aneurysm/dissection, hypertension) as these patients are also at risk for these conditions.

Congenital heart disease is an inherent component of Noonan syndrome. Though a clinical study in Noonan syndrome reported no evidence of somatropin-induced ventricular hypertrophy or exacerbation of preexisting ventricular hypertrophy (as judged by echocardiography), the safety of Norditropin® in children with Noonan syndrome and significant cardiac disease is not known. Cases of pancreatitis have been reported rarely in children and adults receiving somatropin treatment, with some evidence supporting a greater risk in children compared with adults. Girls who have Turner syndrome may be at greater risk than other somatropin-treated children. Pancreatitis should be considered in any somatropin-treated patient, especially a child, who develops abdominal pain.

Other somatropin-related adverse reactions include injection site reactions/rashes, lipatrophy and headaches. Subcutaneous injection of somatropin at the same site repeatedly may result in tissue atrophy and can be avoided by rotating the injection site. Somatropin inhibits 11B-hydroxysteroid dehydrogenase type 1 (11B-HSD-1) in adipose/hepatic tissue, and may significantly impact the metabolism of cortisol and cortisone. In patients treated with somatropin, previously undiagnosed central (secondary) hypoadrenalism may be unmasked requiring glucocorticoid replacement therapy. In addition, patients treated with glucocorticoid replacement therapy, especially with cortisone acetate and prednisone, for previously diagnosed hypoadrenalism may require an increase in their maintenance or stress doses. Careful monitoring is advisable when somatropin is administered in combination with other drugs known to be metabolized by CYP450 liver enzymes (e.g., corticosteroids, sex steroids, anticonvulsants, cyclosporine) as limited published data suggest somatropin may alter clearance of these compounds.

In adult women on oral estrogen replacement, a larger dose of somatropin may be required to achieve the defined treatment goal. The safety and effectiveness of Norditropin® in patients age 65 years and older has not been evaluated in clinical studies. Elderly patients may be more sensitive to the actions of somatropin and may be more prone to develop adverse reactions.

Please see Brief Summary of Prescribing Information on the following pages.