UWHealth

Very Severe Hypertriglyceridemia (TGL ≥ 1000 mg/dL): Management - Adult - Inpatient/Ambulatory/Emergency Department - Clinical Practice Guideline

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Introduction

Hypertriglyceridemia is a common dyslipidemia that has many causes.[1] It is associated with increased atherosclerotic cardiovascular disease (ASCVD) risk that often coexists with other lipoprotein abnormalities.[2] Mild and moderate hypertriglyceridemia places individuals at increased ASCVD risk, but severe and very severe hypertriglyceridemia places individuals at additional risk of pancreatitis and chylomicronemia syndrome.[3] This guideline outlines treatment of severe-very severe hypertriglyceridemia. "Very severe" is defined for the focus of this guideline and to answer the clinical questions outlined below. For management of mild-moderate, or severe hypertriglyceridemia, refer to the UW Health Hyperlipidemia: Management – Adult – Inpatient/Ambulatory clinical practice guideline for ASCVD risk reduction.

Scope

Intended User(s): Physicians, Advanced Practice Providers, Registered Nurses, Pharmacists

Objective(s): To provide guidance on treatment of very severe hypertriglyceridemia for patients seen in the emergency room, urgent care, or who are admitted.

Target Population: Adult patients (age ≥ 18 years) with very severe hypertriglyceridemia (triglycerides ≥1000 mg/dL)

Clinical Questions Considered:

- Which patients with very high triglycerides should be admitted for inpatient care?
- What are the initial diagnostic strategies for patients with very severe hypertriglyceridemia?
- What are the treatment options for patients with very severe hypertriglyceridemia?
- When is plasmapheresis indicated for patients with hypertriglyceridemia?
- What lifestyle and nutrition therapies are recommended for patients with hypertriglyceridemia?

Definitions

Table 1. Categories of Triglycerides Elevations[3-5]

Normal	< 150 mg/dL
Mild	150-199 mg/dL
Moderate	200-499 mg/dL
Severe	500-999 mg/dL
Very severe	≥1000 mg/dL

Causes of Hypertriglyceridemia

Hypertriglyceridemia can be primary or secondary (i.e., due to other conditions and treatments. See **Table 2**)[1] Triglycerides are generally measured in the fasting state and can be divided into categories: normal, mild, moderate and severe elevations (see **Table 1**). Hypertriglyceridemia can result from increased triglycerides production, decreased catabolism, or a combination of both.[3] Almost all patients with very severe elevations in triglycerides have primary and secondary contributors.[1]

Table 2. Causes of Hypertriglyceridemia[1]

Primary Causes	Secondary Causes
• Familial combined hyperlipidemia • Lipoprotein lipase deficiency • Familial dysbetalipoproteinemia • Apolipoprotein CII deficiency • Apolipoprotein C-III excess • Familial chylomicronemia syndrome	• Untreated/poorly controlled diabetes mellitus • Obesity • High fat/high carbohydrate/high caloric diet • Excessive alcohol consumption • Hypothyroidism • Nephrotic syndrome • Pregnancy • Medications (see Table 3)

Metabolism

In the exogenous pathway, dietary triglycerides (fats) are assembled in the intestinal wall into chylomicrons which enter the venous circulation. Through interactions with lipoprotein lipase on the luminal surface of the capillary endothelial cells, triglycerides in the core of the chylomicrons are hydrolyzed into free fatty acids and glycerol which can be used as energy sources or for gluconeogenesis.[3] In the endogenous pathway, triglycerides produced by the liver are carried by very low-density lipoproteins (VLDL) and are also catabolized by lipoprotein lipase. The plasma triglycerides level reflects the concentration of triglyceride-carrying lipoproteins (mainly VLDL and chylomicrons). In severe and very severe hypertriglyceridemia, the lipoprotein lipase catabolism system is saturated. Thus after a fatty meal, when a patient's level is already above 1000 mg/dL, the triglycerides level can significantly increase.[6]

Clinical Presentation

Severe and very severe hypertriglyceridemia may result in hyper-viscous serum and a clinical condition called "chylomicron syndrome" which manifests as acute pancreatitis, abdominal pain, impaired cognition/memory, paresthesias, and hepatosplenomegaly.[3,7] Hypertriglyceridemia-induced pancreatitis was the third leading cause of pancreatitis (~10% of cases) in a population based study.[8] Assessment of the patient who presents with severe/very severe triglycerides elevation includes a careful medical history, clinical exam, laboratory assessment, and evidence chylomicron syndrome symptoms (e.g., paresthesias, abdominal pain.) Because there is no specific triglycerides level that will always result in chylomicron syndrome manifestation, a careful medical history must be part of the clinical assessment.

When the serum triglycerides level exceeds 1000 mg/dL, typical physical exam findings can be found. Eruptive xanthomas are skin manifestations of very severe triglycerides elevations due to trapping of triglycerides in cutaneous histiocytes. Eruptive xanthomas are yellow papules on an erythematous base found on the extensor surfaces of the extremities and on the buttocks.[7] In addition, those with type III hyperlipoproteinemia can have striate xanthomata of the palms tuberoeruptive xanthoma. When triglycerides are high, the serum can have a milky supernatant indicating the presence of chylomicrons. Lipemia retinalis (pallor of the optic fundus where the retinal veins and arteries appear white or creamy) also may be present.[9]

Recommendations

Clinical Assessment

The medical history should focus on assessment of symptoms (see **Figure 2, Pathway Diagram**). These symptoms can be as overt as acute pancreatitis and/or as non-specific as abdominal pain, nausea/vomiting, vision changes, impaired cognition, or paresthesias.[7] A thorough search for contributing medications (see **Table 3**) or secondary conditions (see **Table 2**) should be performed. Laboratory data such as a serum glucose, hemoglobin A_1C, creatinine, thyroid stimulating hormone, urinalysis with protein: creatinine ratio should be obtained in patients that present with severe/very severe hypertriglyceridemia *(UW Health Low quality of evidence, C recommendation)*. Please note that these triglyceride-rich lipoproteins may also result in falsely low amylase levels and pseudohyponatremia.[9,10] In one study, serum amylase was near-normal in 50% of patients presenting with hypertriglyceridemia-induced pancreatitis, therefore if clinical suspicion remains elevated, appropriate imaging may need to be obtained.[7,10]

Table 3. Common drugs/medication that can increase triglycerides[11]

- Beta-blockers
- Glucocorticoids
- Estrogens
- Progestins
- Tamoxifen
- Androgenic steroids
- Retinoids, isotretinoin
- Thiazide/thiazide-type diuretics
- Loop diuretics
- Alcohol
- Protease inhibitors
- Tacrolimus
- Cyclosporine
- Clozapine
- Atypical antipsychotics (e.g., olanzapine)
- Valproate

***Other medications also may contribute*

Acute Management of Very Severe Hypertriglyceridemia

Acute management of very severe hypertriglyceridemia (>1000 mg/dL) is based on symptoms. If the patient has pancreatitis or hyperviscosity symptoms such as abdominal pain, nausea/vomiting, vision changes, impaired cognition or paresthesias, the patient should be admitted to the hospital for more definitive treatment. If the patient is asymptomatic, then secondary or reversible causes should be addressed based on initial laboratory and clinical work-up.

Fenofibrate (160 mg/day) or micronized fenofibrate (200 mg/day) should be started if therapy is not contraindicated, patient's creatinine clearance >30 ml/min, and liver enzymes are acceptable (i.e., AST/ALT <2.5x ULN). [12] *(UW Health Low quality of evidence, C recommendation)* Heparin is not routinely recommended for treatment of severe hypertriglyceridemia due to bleeding risk, especially if the patient has pancreatitis, unless there is a strong indication for use (e.g., pulmonary embolism, deep vein thrombosis, etc.) [13] *(UW Health Low quality of evidence, C recommendation)*

The patient will need close follow-up appointments and it is suggested to follow-up with a primary care physician within 2 weeks and preventive cardiology within 6 weeks. *(UW Health Very low quality of evidence, C recommendation).* Patient should not be discharged until the serum triglycerides are under 500 mg/dL and patient is clinically stable *(UW Health Very low quality of evidence, C recommendation).*

Symptomatic Patient with Very Severe Hypertriglyceridemia

The patient with symptomatic hypertriglyceridemia should be admitted to the hospital and be made N.P.O. to allow for clearance of chylomicrons.[3] *(UW Health Low quality of evidence, C recommendation)* Secondary causes should be identified and addressed. Offending medications should be stopped. A nutrition consult should be placed for all patients with very severe hypertriglyceridemia.[3] *(UW Health Low quality of evidence, C recommendation)* Once the serum triglycerides are <1000 mg/dL, the patient should be started on a very low-fat diet (< 15% of calories), calorically appropriate diet. *(UW Health Low quality of evidence, C recommendation)*

Hyperglycemia should be addressed through glycemic control (e.g., metformin, insulin infusion, diabetes management consultation) and triglycerides-lowering pharmacotherapy should be started. In the setting of concurrent hyperglycemia with hypertriglyceridemia, intravenous insulin may be considered for glucose and triglycerides control. [14,15]*(UW Health Low quality of evidence, C recommendation)*

Fibrates are the first line treatment of hypertriglyceridemia if not contraindicated. If creatinine clearance is >30 ml/min, and AST/ALT <2.5x ULN, fenofibrate (160 mg/day) or micronized fenofibrate (200 mg/day) should be started.[3,12] *(UW Health Low quality of evidence, C recommendation)* In general, niacin is not used.[3] Statins and high dose omega-3 fatty acid containing fish oil capsules are adjunctive therapies once triglycerides are <1000 mg/dL. [11,16] *(UW Health Low quality of evidence, C recommendation)* For patients without pancreatitis, once diet is tolerated and symptoms resolve, the patient should be scheduled for close follow up with the primary care provider (2 weeks) and preventive cardiology (6 weeks). *(UW Health Low quality of evidence, C recommendation)*

Plasmapheresis

In patients with hypertriglyceridemia-induced pancreatitis or severe hyperviscosity symptoms, plasmapheresis can lower triglycerides and lead to symptom resolution more quickly than standard medical care; it also can reduce length of stay.[12,18] However, the effects of therapeutic plasmapheresis for hypertriglyceridemia-induced pancreatitis on clinical outcomes have not been demonstrated in randomized clinical trials.[12] In a small single center case series there was an 80% reduction in plasma triglycerides with the use of therapeutic plasma exchange, but there was no difference between early and late mortality.[18] Because patients with hypertriglyceridemia-induced pancreatitis are at increased risk of mortality (up to 30% in some series)[17] plasmapheresis should be considered in patients with pancreatitis, abdominal pain, or hyperviscosity symptoms *(UW Health Low quality evidence, C recommendation)*.

Patients receiving plasmapheresis uncommonly can have a reaction characterized by nausea, flushing, and hypotension, so the decision to perform plasmapheresis and its timing must be considered with consultation from the Pathology and Laboratory Medicine Transfusion Service.

Patients on ACE inhibitors (ACE-I) who are treated with plasmapheresis can uncommonly develop neurochemical hypotension that may be severe and life threatening. One proposed mechanism of this phenomenon is that ACE inhibitors prolong the half-life of bradykinin. The decision to perform plasmapheresis, and its timing with respect to ACE-I, must be considered in consultation with transfusion medicine physicians (see **Table 5**).

- ACE-inhibitors should be discontinued, when possible, for at least 24 hours prior to and until plasma exchanges have been completed.[19,20] *(UW Health Low quality evidence, C recommendation)*

- If an ACE-inhibitor has been administered within the past 24 hours, plasmapheresis usually is deferred unless the potential benefit outweighs the potential excess risk. *(UW Health Low quality evidence, C recommendation)*
  - These risks cannot be quantified so decision-making should be on a case-by-case basis, have multidisciplinary input, and should be shared with the patient so they can provide informed consent.
  - If an ACE-inhibitor has been administered within the past 24 hours, plasmapheresis must be performed in an intermediate medical care (IMC) or intensive care unit (ICU) setting.[19,20]
- It generally is safe to defer plasmapheresis for hyperviscosity syndrome due to very severe hypertriglyceridemia if the patient has received an ACE-inhibitor in the past 24 hours. *(UW Health Low quality evidence, C recommendation)*
- For patients with abdominal pain or evidence of pancreatitis by labs or imaging, assess for signs of systemic inflammatory response syndrome (SIRS) (see **Table 4**). The presence of 2 or more signs of SIRS may identify patients in whom earlier plasmapheresis might be useful.[21] *(UW Health Low quality evidence, C recommendation)*

Table 4. Signs of SIRS[21]

SIRS- defined by presence of two or more criteria:

- Heart rate >90 bpm
- Core temperature <36°C or >38°C
- White blood count <4,000 or >12,000/mm^3
- Respirations >20/min or $PC0_2$ <32 mm Hg[13]

Table 5. Considerations for Plasmapheresis in Patients with Pancreatitis or Abdomen Pain Due to Very-High Triglycerides

ACE-I Usage	SIRS ≥ 2	SIRS = 0 or 1
No ACE-I taken in past 24 hours	Favors performing plasmapheresis	Consider plasmapheresis
ACE-I taken within past 24 hours*	Consider plasmapheresis with extra caution	Favors deferring plasmapheresis

**If plasmapheresis is performed within 24 hours of the last ACE inhibitor dose, it must be done in an IMC or ICU setting, only after multidisciplinary consultation and patient informed consent. The primary medical service may need to assist with the informed consent process.*

Consult Interventional Radiology if central line placement is required. The Cardiology Consultation team and any of the Preventive Cardiology faculty are available as resources, though formal consultation is usually not necessary.

Disclaimer
Clinical practice guidelines assist clinicians by providing a framework for the evaluation and treatment of patients. This guideline outlines the preferred approach for most patients. It is not intended to replace a clinician's judgment or to establish a protocol for all patients. It is understood that some patients will not fit the clinical condition contemplated by a guideline and that a guideline will rarely establish the only appropriate approach to a problem.

Methodology

Development Process
Each guideline is reviewed and updated a minimum of every 3 years. All guidelines are developed using the guiding principles, standard processes, and styling outlined in the UW Health Clinical Practice Guideline Resource Guide. This includes expectations for workgroup composition and recruitment strategies, disclosure and management of conflict of interest for participating workgroup members, literature review techniques, evidence grading resources, required approval bodies, and suggestions for communication and implementation.

Methods Used to Collect the Evidence:
The following criteria were used by the guideline author(s) and workgroup members to conduct electronic database searches in the collection of evidence for review.

Literature Sources:
- Hand-searching journals, external guidelines, and conference publications

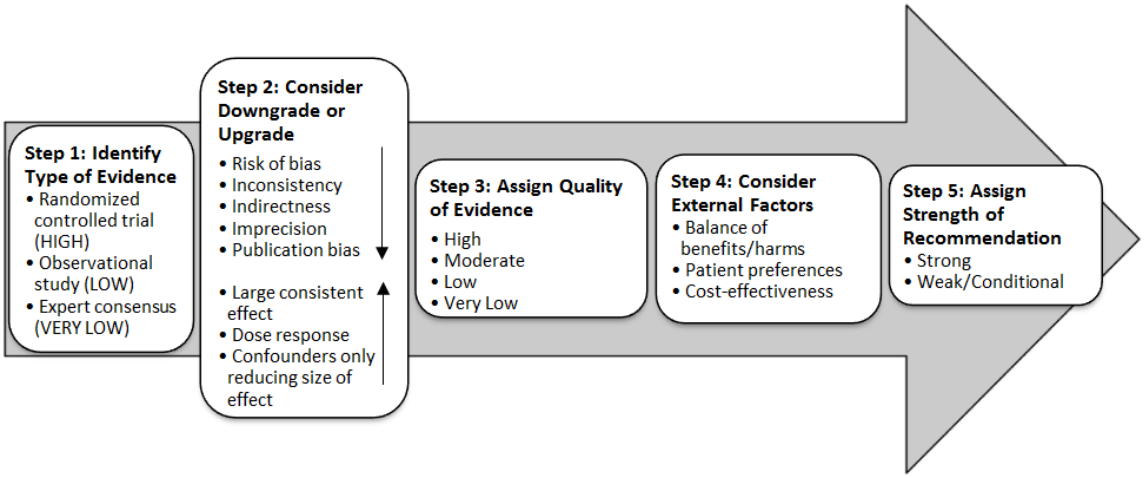
Time Period: 2017-2018

Methods to Select the Evidence:
English journals, leading medical journals, professional society publication.

Methods Used to Formulate the Recommendations:
The workgroup members agreed to adopt recommendations developed by external organizations and/or created recommendations internally via a consensus process using discussion of the literature and expert experience/opinion. If issues or controversies arose where consensus could not be reached, the topic was escalated appropriately per the guiding principles outlined in the UW Health Clinical Practice Guideline Resource Guide.

Methods Used to Assess the Quality of the Evidence/Strength of the Recommendations:
Internally developed recommendations, or those adopted from external sources without an assigned evidence grade, were evaluated by the guideline workgroup using an algorithm adapted from the Grading of Recommendations Assessment, Development and Evaluation (GRADE) methodology (see **Figure 1**).

Figure 1. GRADE Methodology adapted by UW Health



Rating Scheme for the Strength of the Evidence/Recommendations:

GRADE Ranking of Evidence

High	We are confident that the effect in the study reflects the actual effect.
Moderate	We are quite confident that the effect in the study is close to the true effect, but it is also possible it is substantially different.
Low	The true effect may differ significantly from the estimate.
Very Low	The true effect is likely to be substantially different from the estimated effect.

GRADE Ratings for Recommendations For or Against Practice

Strong (S)	Generally should be performed (i.e., the net benefit of the treatment is clear, patient values and circumstances are unlikely to affect the decision.)
Conditional (C)	May be reasonable to perform (i.e., may be conditional upon patient values and preferences, the resources available, or the setting in which the intervention will be implemented.)

Recognition of Potential Health Care Disparities: None identified.

Collateral Tools & Resources

The following collateral tool and resource support staff execution and performance of the evidence-based guideline recommendations in everyday clinical practice.

Metrics

- # of patients admitted with very severe hypertriglyceridemia
- # of ER visits with very severe hypertriglyceridemia

Related Guidelines

Preventive Health Care – Adult/Pediatric – Ambulatory
Hyperlipidemia: Management – Adult – Inpatient/Ambulatory

Patient Resources

1. HFFY #519: Food Guidelines to Reduce LDL Cholesterol and Triglycerides
2. HFFY #5419: Heart Healthy Living for Women
3. HFFY #5668: A Health Guide for Women 50 or Older
4. HFFY #5669: A Health Guide for Men 50 or Older
5. HFFY #6196: Improving Your Lipid (Cholesterol) Level
6. HFFY #6419: A Health Guide for Men 50 or Older
7. HFFY #7466: Familial Hypercholesterolemia (FH) in Children
8. HFFY #7617: My Child's Lipoprotein (a) Level
9. HFFY #7739: Your Risk of Heart and Vascular Disease
10. HFFY #7979 Getting Ready for your Fasting Blood Draw
11. Healthwise: Cholesterol and Triglycerides Tests
12. Healthwise: Cholesterol and Triglycerides Tests: Pediatric
13. Healthwise: Cholesterol and Triglycerides Tests: Teen
14. Healthwise: Well Visit: 18 to 50 Years
15. Healthwise: Well Visit: 50 to 65 Year Men
16. Healthwise: Well Visit: 50 to 65 Year Women
17. Healthwise: Well Visit: Over 65 Years
18. Health Information: Cholesterol in Children and Teens
19. Health Information: Lipid Panel

Smartset

Hyperlipidemia [87]

Protocols

Laboratory Screening and Chronic Disease Monitoring Laboratory Test Ordering in Primary Care – Adult/Pediatric – Ambulatory [93]
Primary Care Lipid Management for Prevention of Atherosclerotic Cardiovascular Disease – Adult – Ambulatory [163]

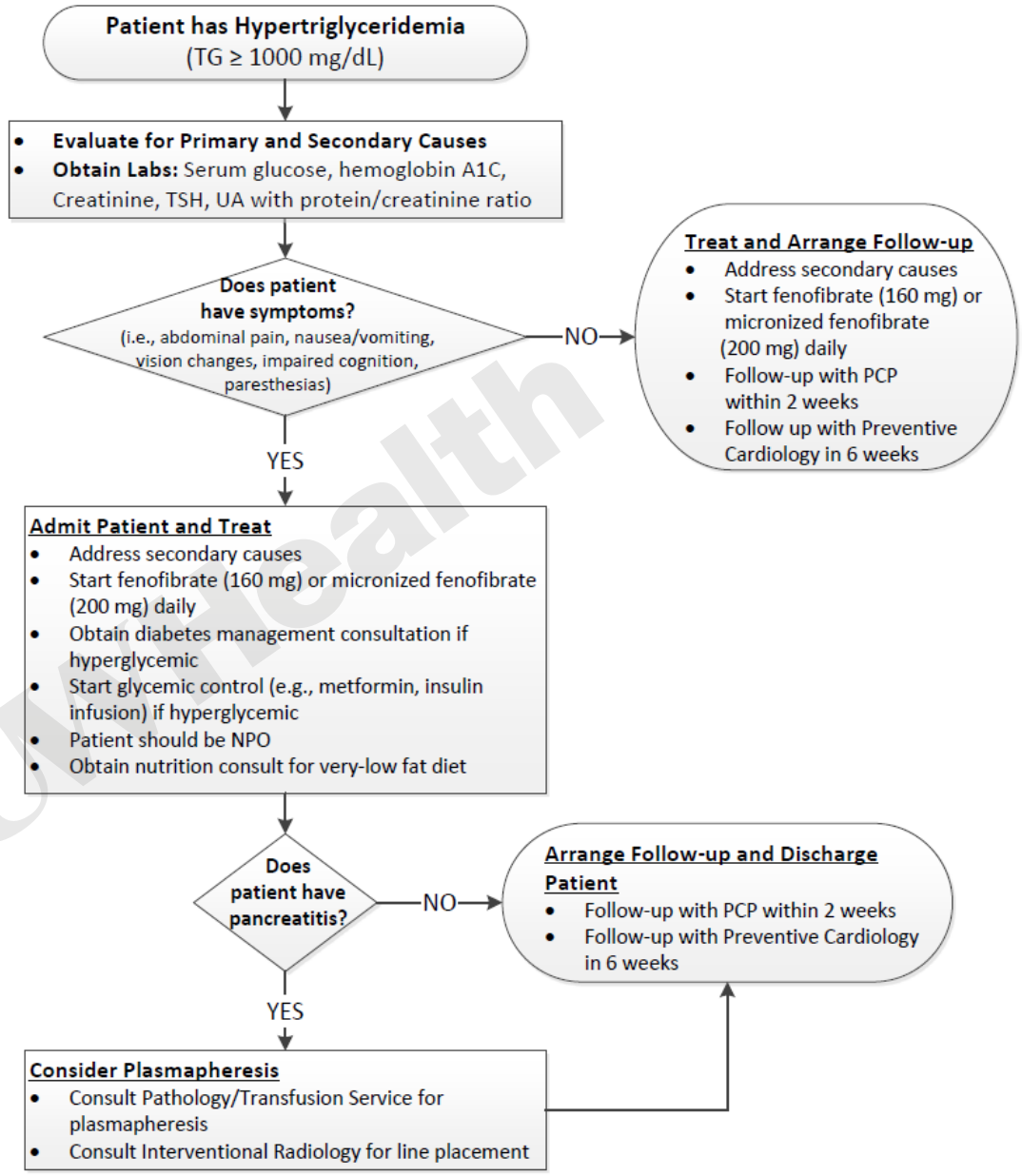
Appendix A. Management of Very Severe Hypertriglyceridemia Algorithm

Primary and Secondary Causes of Hypertriglyceridemia	
Primary Causes	**Secondary Causes**
• Familial combined hyperlipidemia • Lipoprotein lipase deficiency • Familial dysbetalipoproteinemia • Apolipoprotein CII deficiency • Apolipoprotein C-III excess • Familial chylomicronemia syndrome	• Untreated/poorly controlled diabetes mellitus • Obesity • High fat/high carbohydrate/high caloric diet • Excessive alcohol consumption • Hypothyroidism • Nephrotic syndrome • Pregnancy • Medications (see table below)

Common drugs/medications that can raise triglycerides	
• β–blockers • Glucocorticoids • Estrogens • Progestins • Tamoxifen • Androgenic steroids • Retinoids, isotretinoin • Protease inhibitors • Thiazide/thiazide-type diuretics	• Loop diuretics • Tacrolimus • Cyclosporine • Clozapine • Isotretinoin • Protease inhibitors • Atypical antipsychotics (e.g., olanzapine) • Valproate • Alcohol

Consideration for Plasmapheresis if ACE-I Usage		
ACE-I usage	**SIRS ≥ 2**	**SIRS = 0 or 1**
No ACE-I taken in past 24 hours	Favors performing plasmapheresis	Consider plasmapheresis
ACE-I taken within past 24 hours*	Consider plasmapheresis with extra caution	Favors deferring plasmapheresis

** If plasmapheresis is performed within 24 hours of last ACE inhibitor dose, must be done in IMC or ICU setting only after multidisciplinary consultation and patient informed consent*



Appendix B. Nutrition Therapy for Hypertriglyceridemia

Although some patients have a genetic predisposition to hypertriglyceridemia (HTG), secondary HTG is more common and can be due to certain medications, endocrine conditions, and lifestyle factors such as a diet high in refined carbohydrates (CHO) and/or saturated fat, excessive alcohol intake, lack of physical activity, and obesity.[1] Besides pharmacologic interventions, lifestyle modification is crucial for long-term management of HTG. Identifying and optimizing nutrition–related practices can reduce TG levels by ≥ 20-50%[2] (**Table 6**). This document outlines non-pharmacologic approaches to lower TG.

Management of Severe and Very Severe HTG with Lifestyle Therapy

Nutrition therapy is different for individuals with severe HTG (>500 mg/dL) than those with mild/moderate HTG (<500 mg/dL) (**Table 7**). When TG levels are >500 mg/dL, and especially >1000mg/dL, the primary clinical objective is to lower the TG concentration to <500 mg/dL to reduce risk of pancreatitis and chylomicron syndrome.

1. Dietary fat: **A very low-fat diet is critical for managing very high HTG** since dietary fat intake causes formation of chylomicrons, which have delayed clearance in patients with severe HTG (>500 mg/dL). A very low-fat diet is defined as <15% total energy intake or total calories to reduce the formation of chylomicron particles thereby preventing chylomicronemia. This correlates to about 33 grams/day based on a 2000 kcal diet (**Table 7**). This restriction of dietary fat to < 15% is especially important in individuals with fasting TG levels of >1000mg/dL.[3] Dietary fat restriction is usually temporary and after chylomicron particles are cleared, restriction can be lifted gradually while monitoring fasting TG levels. However, in patients with lipoprotein lipase deficiency, dietary fat restriction for an extended period of time may be necessary.[4]

2. Medium chain triglyceride (MCT): Patients on very low-fat diets (<15% total calories) for an extended period of time may benefit from MCT oil if meeting caloric needs is challenging in the initial phase of clearing chylomicrons. MCTs are directly absorbed into portal circulation and do not induce chylomicron synthesis.[5]

3. Omega-3 fat supplementation: Intakes of 2 to 4 grams/day of long-chain omega-3 fatty acids such as eicosapentanoic acid (EPA) and docosahexaenoic acid (DHA) can reduce TG levels by 20-50% in patients with elevated TG.[6] Intake of supplemental long-chain omega-3 fatty acids at therapeutic dosages of 2 to 4 grams/day should be under the supervision of physician or qualified clinician. For patients on a very low-fat diet for an extended period of time, consider providing fats high in monounsaturated fatty acids (MUFA) and polyunsaturated fatty acids (PUFA) that are within recommended amounts from sunflower, olive, avocado, or walnut oils (**Table 9**). Prescription EPA and EPA+DHA concentrates are indicated for the treatment of HTG >500 mg/dL but may not be absorbed well on an empty stomach or when consumed with low-fat meals,[7] so it is important to provide some fat in the diet. In some cases, fat-soluble vitamin supplementation may be necessary to prevent deficiency and therefore continued monitoring is important.

4. Dietary long chain omega-3 fatty acids (marine-derived): Although individuals with severe HTG benefit from omega-3 supplements and not necessarily from dietary fish intake, two servings of fatty fish intake as a part of an extremely low-fat diet will provide the necessary polyunsaturated fats and protein without increasing carbohydrates in the initial phase of chylomicron clearance. Estimates of EPA and DHA content from fish high in long chain omega-3 fatty acids are shown in **Table 10**.

5. Alcohol: Alcohol leads to HTG and weight gain. Alcohol intake should be avoided in people with severe HTG (>500 mg/dL) as it can increase risk of pancreatitis.[2] Alcohol intake should be limited in people with mild (150-199 mg/dL) or moderate (200-499 mg/dL) HTG. The mechanisms via which alcohol induces hypertriglyceridemia include increased very low-density lipoprotein (VLDL) secretion, impaired lipolysis, and increased hepatic delivery of free fatty acid from adipose tissue.[8] Population studies show linear association with alcohol intake and plasma TG concentration.[9,10] Ingestion of

1 oz/day (30g) of alcohol can increase TG concentration by 5% to 10% when compared to nondrinkers.[11] Obesity further exacerbates alcohol-induced HTG especially when accompanied with high saturated fat diet.[8] Individuals with TG >500 mg/dL should be advised to abstain completely from alcohol intake. **Table 11** provides estimates of alcohol content in standard drinks per the National Institutes on Alcohol Abuse and Alcoholism.[12]

6. Fructose: Consumption of foods including beverages high in added sugar and refined carbohydrates has increased significantly in the past 3-4 decades, in parallel with the obesity epidemic. Food and beverages sweetened with "added sugar" contain 50% fructose; food and beverages sweetened with high fructose corn syrup (HFCS) contain 42-55% fructose. Fructose metabolism in the liver is not regulated or inhibited and promotes TG synthesis. Fructose intake is associated with HTG especially when there is positive energy balance and excess intake.[13] Excessive fructose intake in the diet (>50-100 grams/day) is associated with dose-related increases in plasma TG levels.[14] Patients with HTG should be encouraged to avoid added sugars, sugar-sweetened beverages, foods and beverages high in HFCS. Fruits and fruit juices high in fructose should be limited. The American Heart Association recommends limiting added sugars (e.g. table sugar, cane sugar) to fewer than 100 calories daily (e.g. 6 tsp) for women and 150 calories daily (9 tsp) for men (~5% of total energy).[15] **Table 12** provides estimated fructose content of selected foods and beverages.

7. Refined carbohydrates and fiber: Substituting ~16% of calories from refined starches and added sugars with a combination of protein and unsaturated fat in people with HTG can reduce TG levels by 18.5%.[16] Patients should be encouraged to limit refined starches and replace them with protein and unsaturated fat, as well as high fiber and whole grain foods. Whole grains are defined as any grain that contains >3 grams of fiber per one-ounce equivalent serving per day.[17] Whole grains include bran, germ, and endosperm. Bran is a rich source of nutrients such as soluble and insoluble fiber, B vitamins, minerals, flavonoids, and tocopherols. Germ is a good source of several healthy fatty acids, antioxidants, and phytochemicals and endosperm constitutes of mostly starch (CHO polysaccharides) and storage proteins.[18] Refined grains are processed in which bran and germ are removed; this lowers nutrient and fiber content of the grain. Remaining part of the grain which includes endosperm increases the glycemic response of the grain due to increased bioavailability and rapid digestion of the starch in the endosperm. Processing also eliminates mineral, micronutrients, and other phytochemicals that have additional independent health benefits.[19]

Management of Mild to Moderate HTG with Lifestyle Therapy

For patients with mild to moderate HTG (150-499 mg/dL), the primary objective of nutrition therapy is to reduce ASCVD risk by lowering levels of atherogenic lipoproteins (**Table 7**).

1. Dietary long chain omega-3 fatty-acids – marine- and plant-derived: For people with mild (150-199 mg/dL) to moderate (200-499 mg/dL) HTG, consumption of ~ 250 to 550 mg/day of marine-derived EPA and DHA can reduce the risk of death from coronary heart disease.[4] This can be achieved with increased fatty fish intake of two servings per week at 4 oz per serving especially in individuals at high-risk and average-risk persons. Farm-raised and wild-caught seafood contain approximately the same amounts of omega-3 fatty acids (**Table 10**).

   Plant-based omega-3 fatty acids (alpha-linolenic acid, ALA) support cardiovascular health when 2–3 grams/day are consumed.[19] However, they are less effective at lowering TG than marine-based omega-3 fatty acids and conversion of ALA to EPA and DHA is very inefficient, so marine-based fatty acids are strongly preferred. Individuals who cannot incorporate marine-based omega-3 fatty-acids in the diet (due to allergies or other reasons) can include plant-based sources of omega-3 such as flaxseed and flaxseed oil, vegetable oils (e.g. soybean oil, canola oil), and some nuts (e.g. walnuts). Intake of 2-3 g/day of ALA intake can be met with 1-ounce (28-g) serving of walnuts (2.6 g of ALA), 1 tablespoon (15 g) whole flaxseeds (2.3 g ALA) intake in mixed dishes or in salads. Soybean oil (1 tbsp – 0.9g ALA); canola oil (1 tbsp – 1.3 g ALA); walnut oil (1 tbsp – 1.4g ALA); flaxseed oil (1 tbsp – 8.5g of ALA) can be included for cooking or as salad dressings to provide adequate amounts of ALA.

2. Weight loss: More than one-third of the U.S. population has obesity and the incidence continues to rise. Another one-third has an overweight body habitus.[20] Dyslipidemia is the principal metabolic comorbid condition associated with excess body fat, often characterized by HTG with decreased high density lipoprotein cholesterol (HDL-C) and high density lipoprotein (HDL) molecule dysfunction. Low density lipoprotein cholesterol (LDL-C) can be normal, high, or low, but within people with overweight or obesity, LDL particles tend to be small, dense, and more atherogenic, even when LDL-C levels are low or normal.[21]

   Lipoprotein lipase (LPL) is an enzyme that hydrolyzes TG's from TG-rich lipoproteins (TRLs) such as chylomicrons and VLDL for uptake by different tissues. Obesity impairs lipolysis of TRLs by reducing LPL expression in adipose tissue[22] and reduces LPL activity in skeletal muscle.[23] Weight loss can markedly reduce fasting and non-fasting TGs by increasing LPL activity,[24] with increased catabolism of TRLs.[25] TG and LDL-C decline with weight loss; HDL-C declines during initial weight loss, but then plateaus.[26] The magnitude of reduction in TG is related to magnitude of weight loss and baseline TG values. For example, higher baseline TG levels demonstrate greater reductions with weight-loss but even a small (3–5%) reduction in body weight can lower TG [27] and 5–10% weight loss can lower TG by approximately 20%.[4]

3. Macronutrient composition of the diet associated with weight loss and TG-lowering: Diets that produce energy reduction consistently in a sustainable manner along with other behavior changes can induce weight loss.[28] Low-CHO diets decrease TGs and increase HDL-C to a greater extent than low-fat diets in the presence weight loss.[29-31] A meta-analysis of 19 studies by the Institute of Medicine compared low-fat, high-CHO diets vs. higher-fat diets and showed that for every 5% decrease in total fat, TG level was predicted to increase by 6% and HDL-C to decrease by 2.2%.[29] Other meta-analyses showed that moderate-fat diet (32.5% to 50% of calories from fat) decreased TG to greater extent than a lower-fat diet (18% to 30% of calories from fat) in those with Type II diabetes mellitus (-24.8 mg/dL) and those without Type II diabetes mellitus (-9.4 mg/dL).[30] Another large meta-analysis showed that iso-caloric replacement of CHO's with saturated fats, MUFA or PUFA decreased TG levels by 1-2%.[31]

   When refined grains and added sugars are replaced with fiber-rich whole grains and other complex CHO foods, such as legumes, TGs will decline. However, CHOs from less refined sources, such as whole grains can raise the TG concentration. Therefore, it is preferred that refined grains and added sugars be replaced with foods high in unsaturated fats, protein, nuts, seeds and limited amounts of fiber-rich whole grains and legumes.[32]

4. Dietary patterns associated with TG-lowering: Dietary patterns play an important role in promoting cardiovascular health. Individuals eat foods which form the basis of a diet pattern and there is mounting evidence on the effectiveness of multiple dietary patterns that improve cardiovascular health.

   - *Dietary Approaches to Stop Hypertension (DASH) diet:* The DASH diet emphasizes fruits, vegetables, and low-fat dairy products. It includes whole grains, poultry, fish, and nuts, is low in saturated fat, red meat, sweets, and sugar-sweetened beverages. It is relatively rich in healthy CHOs (55%), has moderate protein (18%) and fat content (27%), and is reduced in saturated fat (<7%), total fat, and cholesterol. The DASH diet meets the major nutrient recommendations, is well-established in its effects on lowering blood pressure and also lowers LDL-C.[33] Partial replacement of CHO in the DASH diet with protein (48% CHO, 25% protein and 27% fat) and unsaturated fats (48% CHO, 15% protein and 37% fat) in the optimum macronutrient intake (OMNI) Heart trial led to reductions in plasma TGs level by ~16% and 9% respectively.[34] The higher protein diet (10% CHO replaced with 10% protein) reduced atherogenic apo C-III–containing LDL and apo C-III–containing VLDL which are TRLs. High protein and unsaturated fat (10% CHO replaced with 10% unsaturated fat) diets reduced plasma total and LDL apo B and produced a lower more metabolically favorable ratio of apo C-III to apo E.[34] Greater reductions in TG with higher protein intake suggests that TG-lowering effect of protein goes beyond just

replacement of CHO**.** These results suggest that substituting protein and unsaturated fats for carbohydrate in the context of a healthy dietary pattern produces less atherogenic lipoprotein profile.[34,35]

To increase protein content of the diet for cardiovascular benefits, plant-based food sources such as nuts, seeds, legumes, seitan, soy and soy products should be emphasized along with adequate amounts of seafood, egg whites and poultry which are good sources of magnesium. To increase unsaturated fat content in the diet, more olive oil, olive oil-based spreads, canola oil, safflower oil should be encouraged. Lower CHO ratio in the diet can be achieved by including limited amount of whole grains (in place of refined carbohydrates) and fruits while incorporating more vegetables for increased potassium content in the diet. Low-fat and fat-free dairy products provide calcium in the diet and should be encouraged. Magnesium, potassium and Calcium are important micronutrients and hallmark of the DASH diet [33] which can be retained by making substitutions mentioned above.[36]

- *The Mediterranean-style diet:* The Mediterranean-style diet pattern emphasizes plant-based foods such as fruits, vegetables, whole grain cereals, legumes, nuts and seeds, includes small amounts of dairy, fish and seafood, animal protein and eggs; wine in moderation (mainly with meals). It is higher in total fat (>35%), typically from olive oil. Red meat, processed meats, and simple and refined CHO are limited.[37] A Mediterranean-style diet reduces TGs by 10-15% more than a low-fat diet.[4] However, recent AHA guidelines concluded that there is low strength of evidence to show consistent effects of the Mediterranean diet on lipid-lowering including TG,[37] in large part because a consistent definition of the diet is lacking and results on the effects of this diet on serum lipids from different studies are inconsistent. The 2013 AHA/ACC guidelines on lifestyle management to reduce cardiovascular risk concluded that current reviews on Mediterranean diet and CVD risk reduction lack methodological quality standards and more research is needed to enhance our understanding of how the Mediterranean diet affects CVD before recommending it for CVD risk reduction, and instead favored the DASH diet.[38]

- *Vegetarian Diets and Soy Protein***:** Consuming soy protein in the amounts of 25 to 50 g/d is both safe and effective at reducing LDL-C by 4-8%.[39,40] One study showed that consumption of 47 g soy protein/day reduced not only serum total-C (-9.3%) and LDL-C (12.9%), but also TG by 10.5%, while increasing HDL-C by 2.4%.[39] One meta-analysis concluded that soy protein intake for 8 weeks in the amount of ~30g/day was associated with LDL-C reduction of 4.2- 5.5%, 3.2% higher HDL-C, and 10.7% lower fasting TG levels.[40] One intervention trial in hypercholesterolemia patients showed that when compared to a milk protein control group, soy protein intake significantly reduced non-HDL-C (6.9%), TG (13.7%), and apo B (7.3%).[41] Increasing soy intake may help displace foods high in saturated fats and cholesterol with foods low in saturated fats thereby improving lipid profile.[42] Soy protein foods are one source of plant protein among others (e.g., nuts, legumes) and can be included as a part of heart healthy diet pattern.

  Data on the benefits of soy in ASCVD prevention are inconclusive, however, soy products such as tofu, soy butter, soy nuts, or some soy burgers are high in polyunsaturated fats, fiber, vitamins, and minerals and low in saturated fat [43] which may be beneficial. Moreover, soy protein could replace other sources of calories such as CHO or fat and can raise the total amount of protein intake while reducing CHO or fat intake. Soy foods that provide 6.25 g of soy protein include 4 oz. of whole soybeans, 8 oz. of soy milk, 3.5 oz. soy flour, 8 oz. textured soy protein, 4 oz. tofu, and 4 oz. tempeh (**Table 13**).

5. Physical Activity and Exercise: Regular exercise has cardio-protective effects mediated by improvements in several lipid-related atherosclerotic risk factors including metabolism of TG-rich lipoproteins. In the absence of weight loss, exercise training reduces TGs and has modest effect on raising HDL-C. In presence of weight loss, TG reduction and increases in HDL-C is further enhanced and LDL-C can be reduced. [44] The magnitude of these changes depends on dietary patterns, type, frequency, intensity, and duration of physical activity, baseline TG values, exercise energy expenditure, and how soon TGs values are measured after the last exercise session. [45]

Mean fasting TG reduction of ~24% (range 4 to 37%) has been noted in intervention trials.[4] Exercise-induced TG-lowering is transient and reverses readily with detraining or when exercise is stopped. Lower fasting plasma TG concentrations after >12 hours of last exercise session wanes after 2 days without exercise suggesting that exercise should be performed consistently and on a regular basis to maintain low TG concentrations.[45] In individuals with high risk of CVD such as obesity and metabolic syndrome and high baseline TG levels, even a small amount of energy expenditure irrespective of type of meal (high fat versus moderate fat) is effective in producing TG reductions similar to those seen in healthy adults.[45]

Higher-intensity interval training (HIIT) exercise (>60% maximum aerobic capacity) produces greater energy expenditure and is more effective at reducing postprandial TG than moderate intensity exercise (resistance training type), though the combination of low-intensity aerobic exercise plus diet (with moderate fat intake, high in MUFA) or resistance training plus aerobic exercise can be equally effective. Exercise in small bouts spread throughout the day has similar benefits on TG metabolism as those seen with continuous exercise.

Summary of Recommendations

Following medical management and stabilization of severe or very-severe HTG, further optimization of nutrition-related practices can help with lowering TGs in a sustainable manner. Nutrition education and counseling should target the following:

1. Increase PUFA and MUFA fats with consumption of marine-based omega-3 fatty acids from fish or supplemental sources.
2. Eliminate industrial trans-fats and restrict saturated fats to 5-6% of total energy intake.
3. Reduce weight if BMI >25 kg/m^2 or increased waist circumference of >102cm in men and >88cm in women.
4. Replace refined carbohydrates with whole grain sources to increase fiber intake and restrict fructose along with other sources of added sugar.
5. Minimize alcohol intake to 1 standard drink per day for women; 1 standard drink per day for men.
6. Implement the Dietary Approaches to Stop Hypertension (DASH) or Mediterranean-style dietary pattern.
7. Increase exercise and physical activity to achieve at least 150 minutes of aerobic exercise each week.

Conclusion

A practical algorithm for lifestyle management of HTG is outlined in **Figure 2**. Overall, the treatment of HTG should focus on therapeutic lifestyle changes after initial medical management and stabilization of complications such as pancreatitis and chylomicronemia syndrome to achieve a decrease in TG of ~50% (**Table 6**). In patients with severe or very severe HTG (>500 mg/dL) or a history of TG-induced pancreatitis, restriction of fat intake to less than 15% total kcals from fat is critical, along with complete abstinence from alcohol. Consider use of MCT oils if it is hard for the patient to meet caloric needs.

A 5-10% reduction in body weight can have an anticipated 20% TG-lowering response. Further offsets in CHO calories may contribute an additional 10% to 20% reduction in TG levels. This should be achieved by reducing added sugars and fructose while increasing unsaturated fat intake. Trans fats should be eliminated and if possible be close to 0% in the diet with restriction in saturated fatty acid. Patients should be advised to increase consumption of marine-based omega-3 products. Aerobic activity along with dietary changes will provide robust TG-lowering effects. Together, these changes can produce reductions of 50% or more in TG levels.

Table 6. Impact of different lifestyle practices on TG lowering

Lifestyle change	TG-lowering response
Weight-loss (5-10%)	20% reduction
Variant of DASH diet: High protein (25%) and High PUFA (37%)	9-16% reduction
Mediterranean style diet pattern versus low-fat diet	10-15% reduction
Omega-3 fatty acids (marine-derived)	~5-10% reduction per gram of omega-3 fatty acids
Decrease refined CHO (1% refined CHO when replaced with 1% PUFA and/or MUFA and protein)	1-2% reduction
Eliminate trans fats	1% reduction
Total TG lowering effect	**~50% or higher reduction**

Table 7. Nutrition Therapy for Hypertriglyceridemia

Nutrition Therapy for High or Very High TGs (>500mg/dL) and Chylomicron Clearing
1. Limit total fat to 10%–15% calories (~33g/day on a 2000 kcal diet). Include oils rich in MUFA and PUFA 2. Complete abstinence from alcohol 3. Avoid refined starches such as white rice, breads, and pastas 4. Avoid potatoes, corn, and other starchy vegetables 5. Replace refined starches with high fiber, whole grain foods partially 6. Avoid foods that have added sugars such as desserts, sugar sweetened beverages and fruit juice and other sugar sweetened juices such as Capri sun, lemonade etc. 7. Limit fruit to 1 serving / day 8. Spread calories and carbohydrates evenly through the day 9. Limit calories if weight loss is indicated. Recommend ~10-15 kcals/kg body depending on baseline body mass index 10. If patient has pancreatitis and has lost a lot of weight and needs extra calories, add medium chain TG oil. Increase oil as needed gradually 11. Once chylomicrons have been cleared and TGs are <500 mg/dL, gradually advance dietary fat intake to tolerance and goal of ~25-35% of total calorie intake, emphasizing omega-3 PUFA sand MUFAs
Nutrition Therapy for High TGs (150-499mg/dL)
1. Total fat can be increased gradually to 25%–35% calories. Include oils rich in MUFA and PUFA 2. Continue to monitor TG levels 3. Very little (0-1 drink a day or less) or moderate alcohol intake (1 drink a day or less) 4. Limit refined starches such as white rice, breads, and pastas. If possible, no more than 1-3 servings a day. 5. Limit potatoes, corn, and other starchy vegetables 6. Replace refined starches with high fiber, whole grain foods 7. Limit foods that have added sugars such as desserts, sugar-sweetened beverages, and fruit juice and other sugar sweetened juices such as Capri sun, lemonade etc. 8. Limit fruit to ~1 or 2 a day 9. Limit calories if weight loss is indicated. Recommend ~10-15 kcals/kg body depending on baseline body mass index 10. Exercise 30–60 min most days. Incorporate moderate to high intensity exercises. Include strength training exercises 1-2 times a week

Table 8. Recommended macronutrient amounts based on total calorie intake and sample of foods from different food groups that can be incorporated in the diet to limit total fat intake for patients with severe or very severe HTG.

Fat (15%)						
	1000 kcal diet	**1200 kcal diet**	**1400 kcal diet**	**1600 kcal diet**	**1800 kcal diet**	**2000 kcal diet**
Grams per day allowed (tsp oil)	17 (~3.5)	20 (4-5)	23 (4.5-5)	27 (6.5)	30 (7.5)	33 (8)
No heavy sautéing and no deep-fried foods Consider Nuts, Seeds, Avocados, Fish for healthy fats Adjust tsp oil used/day based on daily consumption of nuts, seeds, avocados, fish and other sources of fat Sample of daily fat intake is shown below						
Oil (Pam spray) or (tsp)	Add minimum or use spray	Add minimum or use spray	Add minimum or use spray	1tsp	1 tsp	1 tsp
Avocado/day (fat in grams)	¼ (7)	¼ (7)	¼ (7)	¼ (7)	¼ (7)	¼ (7)
Nuts (ounces)/day Almonds (count) (fat in grams) OR Walnuts (Count in halves) (fat in grams)	½ 12 (7.5) 0	½ 12 (7.5) 0	½ 12 (7.5) 0	½ 12 (7.5) 0	½ 0 7 (9)	½ 0 7 (9)
Seeds Sunflower, dried and toasted without salt (ounces) (fat in grams)	0	0	0	0	0	¼ (4)
Fish, salmon, chinook, raw (ounces) (Fat in grams)*	0	1.5 (4.5)	3 (9)	3 (9)	3 (9)	3 (9)
CHO (45-55%)						
	1000 kcal diet	**1200 kcal diet**	**1400 kcal diet**	**1600 kcal diet**	**1800 kcal diet**	**2000 kcal diet**
Grams/day	112-137	135-165	157-192	180-220	202-247	225-275
Grams/meal	37 – 45	45 - 55	50- 64	60- 73	67- 82	75-91
Grams/snack ***Adjust CHO (g) per meal to stay within total recommended amount per day***	15 or less	15 or less	15 or less	15 or less	15 or less	15 or less
No sugar sweetened beverages No added sugar Switch from refined carbs to whole grains Replace whole grains partially Include non-starchy vegetables only See sample plan below, make adjustments as needed						

Whole grains - All whole-grain products and whole grains used as ingredients: E.g. whole-wheat bread, whole-grain cereals and crackers, oatmeal, quinoa, popcorn, and brown rice	3 ounces	4 ounces	5 ounces	5 ounces	6 ounces	6 ounces
Whole grain servings defined:	1 ounce-equivalent is: ½ cup cooked rice, pasta, or cereal 1 ounce dry pasta or rice 1 medium (1 ounce) slice bread 1 ounce of ready-to-eat cereal (about 1 cup of flaked cereal)					
Fruits (Cups)	½	½	1	1	1	1.5
Non-Starchy Vegetables (Cups)	2	3	3	4	5	5
Fruits and Vegetables servings defined:	1 cup-equivalent is: 1 cup raw or cooked vegetable or fruit, 2 cups leafy salad greens, ½ cup dried fruit or vegetable.					
Protein (30-40%)						
	1000 kcal diet	**1200 kcal diet**	**1400 kcal diet**	**1600 kcal diet**	**1800 kcal diet**	**2000 kcal diet**
Grams per day	75-100	90-120	105-140	120-160	135-180	150-200
Eggs (Avoid egg yolk), skinless chicken and turkey. No red meat. Consider adding soy products, legumes (beans and peas), and lentils to increase protein without increasing fat.						
Fish/Seafood (ounces) – Same as above*	0	1.5	3	3	3	3
Chicken/Turkey (ounces) (Lean portions)	0	0	1.5	1.5	2	2
Lentils/Beans/Peas/Legumes (cups)	1	1	1	2	3	3
Soy products (ounces)	3-4	3-4	4-5	5-6	5-6	6-7
Dairy products (Servings) (Only fat-free or very low-fat options) - Consider Fairlife milk (Low-carb and high protein) - Low-fat or fat-free cheese (E.g. Lifetime) - Fat-free and sugar-free Greek or plain yogurt	1	1	1.5	1.5	3	3
Servings Defined	1 ounce lean meat, poultry, or seafood 1 egg ¼ cup cooked beans or tofu 1 cup milk, yogurt 1½ ounces natural cheese such as cheddar cheese or 2 ounces of processed cheese					

*Can replace nuts, based on patient preference. Nutrient values of food from U.S. Department of Agriculture, Agricultural Research Service. Food Data Central, 2019. fdc.nal.usda.gov. Note: Total fat content can be liberalized with monitoring when TG levels reach <500mg/dL.

Table 9. Oils high in polyunsaturated and monounsaturated fats in the diet

	Monounsaturated fat (MUFA) (%)	Polyunsaturated fats (PUFA (%)		
	(Oleic Acid – 18:1)	Linoleic (18:n-6)	Linolenic (18:3n-3)	Total % PUFA
Canola oil	28	19	9	28
Corn oil	28	53	1	54-55
Olive oil	73	10	1	10-11
Peanut oil	46	32	0	32
Safflower oil* (high Linoleic)	14	75	0	75
Safflower oil* (High Oleic)	75	13	1	13-14
Soybean oil	23	50	7	57-58
Sunflower oil (high Linoleic)	20	66	0	66
Sunflower oil* (High Oleic)	84	4	0	4
Avocado oil	70	13	1	14
Walnut oil	23	0	10	63
Walnut oil has 53% 18:2 fatty acids and 10% 18:3 fatty acids making it a total of 63% PUFA				

*Primary oils of commerce

From: U.S. Department of Agriculture, Agricultural Research Service. Food Data Central, 2019. fdc.nal.usda.gov.

Table 10. Polyunsaturated fatty acid composition of common seafood varieties (mg/4ounces)
A total of 1,750 mg of Eicosapentaenoic (EPA) and Docosahexaenoic (DHA) per week represents an average of 250 mg per day, which is the goal amount to achieve at the recommended 8 ounces of seafood per week for the general public.

Salmon: Atlantic, Chinook, Coho	1200-2400
Anchovies, herring and shad	2300-2400
Mackerel: Atlantic and Pacific (not king)	1350-2100
Tuna: Bluefin and Albacore	1700
Sardines: Atlantic and Pacific	1100-1600
Oysters: Pacific	1550
Trout: Freshwater	1000-1100
Tuna: white (albacore) canned	1000
Mussels: blue	900
Salmon: Pink and Sockeye	700-900
Squid	750
Pollock: Atlantic and walleye	600
Eastern oysters	500-550
Spiny Lobsters	550
Crab: blue, king, snow, queen and dungeness	200-550
Tuna: Skipjack and yellowfin	150-350
Flounder, plaice and sole	350
Clams	200-300
Tuna: light canned	150-300
Catfish	100-250
Cod: Atlantic and Pacific	200
Scallops: bay and sea	200
Haddock and hake	200
Lobsters: Northern and American	200
Crayfish	200
Tilapia	150
Shrimp	100
Low in Mercury and Safe in pregnancy	
Shark	1250
Tilefish: Gulf of Mexico	1000
Swordfish	1000
Mackerel: King	450

From: U.S. Department of Agriculture and U.S. Department of Health and Human Services. Dietary Guidelines for Americans, 2010. 7th Edition, Washington, DC: U.S. Government Printing Office, December 2010.

Table 11. Alcohol content of standard drinks

Standard drink	Alcohol by volume (ABV)
12 ounces beer	5%
8 ounces of Malt Liquor	7%
5 ounces of wine but 4 ounces wine is standard drink per American Heart Association	12%
1.5 ounces of 80% proof spirit	40%
1 ounce of 100% proof spirit	50%
Per National Institute on Alcohol Abuse and Alcoholism (NIAAA), a standard drink contains around 14 grams (0.6 ounces) of pure alcohol. Not all alcoholic beverages meet the criteria to be considered a standard drink.	

Source: National Institutes on Alcohol Abuse and Alcoholism. https://www.niaaa.nih.gov/what-standard-drink

Table 12. Fructose content of selected foods and beverages

Product	Amount	Fructose content
Lemonade frozen concentrate white	12 ounces	78.8 g
Regular carbonated cola	16 ounces	28.7 g
Carbonated, ginger ale	16 ounces	18.1 g
Orange juice, chilled, includes from concentrate, with added calcium	12 ounces	5.55 g
Molasses	1 tbsp.	2.6 g
Raisins, golden, seedless	1 ounce (30 g)	10.4 g
POWER BAR, chocolate	1 bar (68 g)	10.9 g
Agave Syrup (Sweetener)	1 tsp	3.84 g
Honey Syrup	1 tbsp.	8.6 g
Sweetened Canned Applesauce	1 ounce (30 g)	2.2 g
Fruit (Banana - raw)	1 medium – 7-8” long – 118 g	5.72 g
Fruit (Pear)	1 medium – 178 g	11.4 g
Table Sugar	1 tsp	2 g

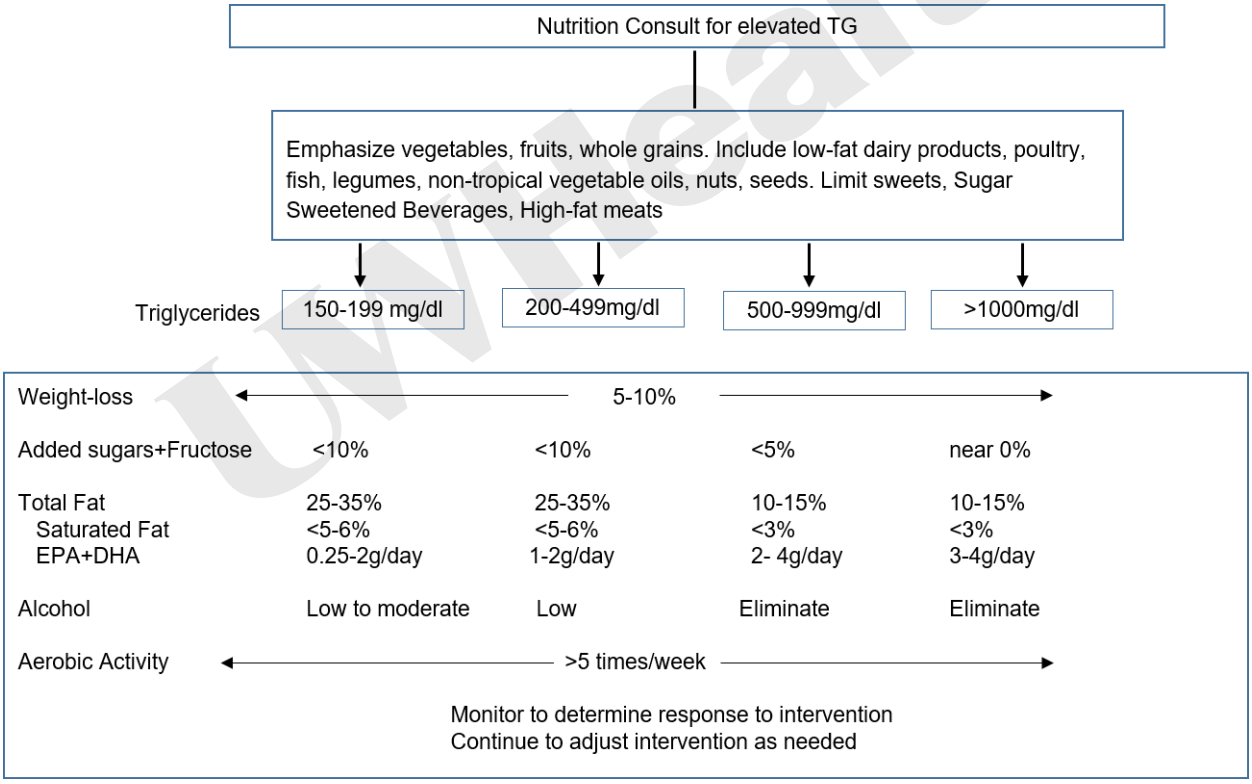
From: U.S. Department of Agriculture, Agricultural Research Service. FoodData Central, 2019. fdc.nal.usda.gov.

Table 13. Soy protein content of popular soy products

Food item	Quantity	Protein in grams
Soy candy bar, chocolate	1 bar (61.5 grams)	14
Soy Burger	1 patty (51 grams)	13
Soy nuts, roasted unsalted	1 ounce (28 grams)	12
Edamame	½ cup (90 grams)	11
Soy hot dog	1 link (42 grams)	9
Tofu extra firm	79 grams	8
Tofu firm	79 grams	7
Soy milk, plain flavor	1 cup, 240 mL	7
Roasted Soy butter	2 tbsp. (32 grams)	6
Soy milk, chocolate	1 cup, 240 mL	5
Tofu Silken	91 grams	4
Miso	2 tbsp. (34 grams)	4

Adapted From: Sacks FM, Lichtenstein A, Van Horn L, Harris W, Kris-Etherton P, Winston M; American Heart Association Nutrition Committee. Soy protein, isoflavones, and cardiovascular health: an American Heart Association Science Advisory for professionals from the Nutrition Committee. Circulation. 2006 Feb 21;113(7):1034-44.

Figure 2. Clinical algorithm for screening and dietary management of elevated TG



Adapted From: Jacobson TA, Maki KC, Orringer CE et al. National Lipid Association Recommendations for Patient-Centered Management of Dyslipidemia: Part 2. J Clin Lipidol. 2015 Nov-Dec;9(6 Suppl):S1-122.

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