

**SEVERE HYPERTRIGLYCERIDEMIA-INDUCED ACUTE PANCREATITIS:
MEDICAL AND NUTRITIONAL IMPLICATIONS**

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EXECUTIVE SUMMARY

Primary hypertriglyceridemia (familial) is caused by genetic defects which lead to errors in triglyceride metabolism (1). Primary hypertriglyceridemia can occur due to a defect in or absence of lipoprotein lipase. Hypertriglyceridemia is typically discovered by a routine lipid profile. A normal triglyceride level is defined as < 150 mg/dL (1, 3). The treatment of hypertriglyceridemia begins with Medical Nutrition Therapy or Therapeutic Lifestyle Changes (TLC). The primary goal of nutritional management is to prevent pancreatitis. If a genetic defect is identified, a very low fat diet ($< 15\%$) is required and patients should be counseled to eliminate all alcohol (5). The goal of treatment for hypertriglyceridemia is to first reduce triglycerides to < 500 mg/dL (1). When the triglyceride level is between 200 and 500 mg/dL, the aim of treatment is to reach goals for: 1) LDL; 2) non-HDL (total cholesterol-HDL); and 3) HDL (> 40 mg/dL).

Acute pancreatitis is an immunoinflammatory response to a localized process of autodigestion of the pancreatic gland. The risk for developing pancreatitis from hypertriglyceridemia becomes clinically significant at levels $> 1,000$ mg/dL. In pregnancy, the incidence of pancreatitis occurs more commonly in the third trimester and postpartum period (7). Amylase and lipase are the most common laboratory markers used to diagnose acute pancreatitis. Enteral nutrition is the recommended method of nutrition support with the enteral tube placed into the distal portion of the duodenum or into the jejunum. Implications of findings to practice of dietetics include:

- Application of the Nutrition Care Process and Nutrition Care Manual in Nutritional management.
- Effectiveness of medical nutrition therapy in nutritional management.
- EN now considered first choice for nutrition support for acute pancreatitis.
- RD plays a key role in nutrition support even when TPN is managed by PharmD.

CASE REPORT

General Information

MJ is a 28 year old African American female who was diagnosed with severe hypertriglyceridemia (1125 mg/dL) in July 2007 during her pregnancy. She presented to Anne Arundel Medical Center (AAMC) with severe, radiating chest pain, shortness of breath, nausea and vomiting. MJ reported a previous episode of the same symptoms after eating a “large meal”. Plan for medical treatment was diet therapy only during the rest of the pregnancy and post-partum due to contraindication of medications and plans for breastfeeding. MJ presented to AAMC again on 9/16/07 at 36 weeks gestation with abdominal pain, nausea and vomiting. After receiving an epidural for pain management, MJ started to have tonic-clonic seizures. The seizure work-up was negative. Once MJ was stabilized, an emergent cesarean section was performed due to fetal distress. MJ delivered a healthy premature female. She was admitted to the Critical Care (CC) Unit for stabilization.

Social History

MJ is married with 1 child and works as a police officer in Anne Arundel County. She is athletic. She denies history of alcohol, smoking, or recreational drugs. She is adopted and does not know her family history.

Medical/Surgical History

PMH: Anemia, Asthma, Severe Hypertriglyceridemia

Nutrition History

Anthropometrics:

Ht.=5' 1" Wt.=155 (70 kg) BMI (Body Mass Index)=30 IBW=105 lbs. % IBW=136

Diet/Physical Activity history: Followed low-fat diet and active athlete prior to admission (PTA).

Estimated energy requirements/day: 1500-1800 kcal (21-26 kcal/kg)

Estimated protein requirements/day: 52-56 gm (0.8-0.9 gm/kg)

Estimated fluid requirements/day: 1500-1800 ml

Hospital Course of Patient

Medical Information:

Pancreatitis secondary to hypertriglyceridemia was diagnosed s/p cesarean section based on elevated amylase (123 mg/dL) and lipase (448 mg/dL). The first CT scan revealed pseudocyst, phlegmon, and no necrosis. A repeat CT scan on 10/31 showed improvement with less fluid and no pseudocysts or necrosis. Intravenous antibiotics were administered due to persistent fevers, nausea, and abdominal pain. MJ attempted breast feeding by pumping her breast milk. However, these attempts were discontinued because of her illness and difficulties with the pump. On 10/05/07, MJ was discharged home NPO and on TPN. She was readmitted on 10/31 with a PICC line sepsis. MJ was treated with intravenous (IV) antibiotics and discharged on home TPN and IV antibiotics.

Nutritional Treatment:

9/20/07

Assessment: MJ was admitted to Critical Care (CC) s/p caesarean section with pancreatitis and hypotriglyceridemia. Patient NPO with nasogastric (NG) suction. Well nourished on admit.

Diagnosis: Altered GI function related to (R/T) ileus as evidenced by (AEB) patient currently NPO with NG suction.

Goal: Adequate kcal and protein intake to prevent malnutrition.

Intervention: Reassess in 3 days. Suggest nutrition support if still NPO.

9/27/07

Assessment: M.J developed an ileus. Total parental nutrition (TPN) initiated by PharmD. Limited nutrition with TPN- no lipids in TPN due to elevated triglycerides. MD plans to keep MJ NPO for at least two more days. Blood sugars adequately controlled.

Diagnosis: Inadequate intake from enteral –parental nutrition infusion R/T hypertriglyceridemia and no lipids in TPN AEB TPN meeting 65% caloric/100% protein needs.

Goal: TPN will meet 100% of calorie and protein needs.

Intervention: Refer to other provider. TPN per PharmD. Advance diet per MD. Suggest dextrose be increased in TPN to better meet nutrition needs.

Monitoring and Evaluation: Lab values, weight, nutrient composition in TPN.

9/30/07

Assessment:

Trial lipids in TPN- monitor lipid clearance. Patient for MRCP to evaluate pancreas and integrity of pancreatic duct. Glucose well-controlled.

Diagnosis: Inadequate intake from enteral –parental nutrition infusion R/T hypertriglyceridemia and trial lipids in TPN AEB TPN meeting 85% caloric/100% protein needs.

Goal: TPN will meet 100% of calorie and protein needs.

Intervention: Refer to other provider. TPN per PharmD. Advance diet per MD. Suggest dextrose be increased in TPN to better meet nutrition needs.

11/01/07

Assessment:

Admitted with fever, chills, nausea, vomiting. Diagnosis: Line sepsis. Antibiotics started. Denied problems with home TPN. Discharged home on intravenous antibiotics. New PIC line inserted right arm.

Diagnosis: Inadequate intake from enteral-parental nutrition infusion R/T abdominal pain, nausea, vomiting, home TPN PTA AEB currently NPO and awaiting restart of TPN.

Goal: TPN will meet 100% of calorie and protein needs once new PICC line inserted and TPN restarted.

Intervention:

Refer to other provider. TPN per PharmD. Advance diet per MD. Continue lipids in TPN. Suggest increase dextrose in TPN to better meet nutritional needs. A low-fat diet is recommended once oral intake is resumed.

CASE DISCUSSION

Medical and Nutritional Considerations/Treatment for Hypertriglyceridemia

There are two types of hypertriglyceridemia- primary or secondary. Primary hypertriglyceridemia (familial) is caused by genetic defects which lead to errors in triglyceride metabolism (1). Primary hypertriglyceridemia can occur due to a defect in or absence of lipoprotein lipase. Lipoprotein lipase releases triglyceride from the chylomicrons. If there is a defect in or absence of lipoprotein lipase, triglyceride is not released from the chylomicrons and triglyceride levels become elevated. MJ was diagnosed with primary (familial) hypertriglyceridemia possibly due to a lipoprotein deficiency. Secondary hypertriglyceridemia may be caused by high fat diets, obesity, diabetes, hypothyroidism, nephrotic syndrome and medications. High carbohydrate diets, certain medications, and alcohol, can also cause or exacerbate hypertriglyceridemia.

The two sources of serum triglyceride are endogenous (from the liver) and exogenous (from dietary fat). Although, the liver produces and secretes a small amount of triglyceride, most of the triglyceride found in the blood is dietary triglyceride which has been absorbed from the small intestine, secreted into the lymphatic system and enters the blood as chylomicrons. Triglyceride is removed from the chylomicrons by muscle and adipose tissue or taken up by the liver and metabolized into a cholesterol-rich lipoprotein (2).

Hypertriglyceridemia is defined as an abnormal level of triglycerides in the blood. According to the National Cholesterol Education program Adult Treatment Panel (NCEP ATP III) guidelines, triglyceride levels can be defined as normal, borderline, high, or very high. A normal triglyceride level is defined as < 150 mg/dL (1,3). Hypertriglyceridemia is typically discovered by a routine lipid profile. In hypertriglyceridemia >500 mg/dL, pancreatitis, eruptive

xanthomas, or lipemia retinalis may also be present (Appendix A). A fasting lipid profile (total cholesterol, low-density lipoprotein, high-density lipoprotein, and triglycerides) should be obtained on patients at age 20 years and repeated every 5 years. In patients who are healthy asymptomatic, and without risk factors, a nonfasting cholesterol and HDL may be obtained every 5 years. In patients with coronary heart disease (CHD) or CHD risk factors, a fasting lipid panel should be obtained yearly. If the triglyceride level is >150 mg/dL, it should be rechecked after a 12-16 hour fast. If the triglyceride level is >1000 mg/dL, beta quantification by ultra centrifugation and electrophoresis can determine the exact dyslipidemia.

Studies have shown an association between hypertriglyceridemia and increased cardiovascular risk. However, hypertriglyceridemia as an independent risk factor for coronary heart disease is controversial (4). In high-risk patients, (those with cardiovascular disease or diabetes), lowering triglyceride levels in addition to lowering LDL cholesterol levels and raising HDL cholesterol levels has been associated with decreased cardiovascular morbidity and mortality. Guidelines from the National Cholesterol Education Program and the American Heart Association have identified triglyceride control and diagnosis of the metabolic syndrome as factors in the management of dyslipidemia (4).

Patients with hypertriglyceridemia should also be evaluated for fasting hyperglycemia, hypertension, abdominal obesity, and low HDL levels for diagnosis of the metabolic syndrome. Thyrotropin level, serum urea nitrogen, creatinine, and urinalysis to evaluate thyroid and renal function and liver function tests should also be assessed. Serum amylase and lipase should be measured for pancreatitis and fasting insulin level for insulin resistance.

The treatment of hypertriglyceridemia begins with Medical Nutrition Therapy or

Therapeutic Lifestyle Changes (TLC). The goals of nutritional management are to prevent pancreatitis and decrease risk for cardiovascular disease. Two nutritional concerns are: 1) decreasing the percentage of carbohydrate and increasing the percentage of fat (up to as much as 35%) may decrease triglycerides, but may also hinder weight loss efforts; and 2) if a genetic defect is identified, a very low fat diet (< 15%) is required and patients should be counseled to eliminate all alcohol (5). Components of TLC include:

1. Counseling on weight management, healthy diet, regular exercise (at least 30 minutes of aerobic exercise 5 days a week)
2. Alcohol and smoking cessation.
3. Total fat 25-35% of total calories
4. Carbohydrates 50-60% of total calories.
5. Protein ~ 15% of total calories
6. Fiber 20 -30 gms. per day
7. Cholesterol < 200 mg/day
8. Avoid sucrose and other simple sugars.
9. Increase intake of oily fish (salmon, mackerel, and herring) to at least two servings a week.
10. Low fat legumes, fruits, vegetables, and nonfat dairy
11. Plant sterols and sterol esters

MJ received counseling from the dietitian on a low-fat diet. Hypertriglyceridemia is very responsive to nutrition therapy. Monitoring and evaluation of the nutrition prescription includes reassessment of current medical condition, relevant lab values, and diet and weight changes. The goal of treatment for hypertriglyceridemia is to first reduce triglycerides to <500 mg/dL (1). When the triglyceride level is between 200 and 500 mg/dL, the aim of treatment is to reach goals for: 1) LDL; 2) non-HDL (total cholesterol-HDL); and 3) HDL (>40 mg/dL). The non-HDL goal is 30 mg/dL higher than the LDL goal. Fibrates, statins, niacin, and fish oil are the main pharmacological agents for managing hypertriglyceridemia. However, MJ did not receive any pharmacological agent because those medications are categorized as Pregnancy Class C and considered potentially harmful to the fetus (Appendix D, 16). Fish oil was considered but it was

not given due to the risk of mercury toxicity. MJ also did not receive medications post-partum due to her attempts at breast feeding.

For isolated hypertriglyceridemia, fibrates, such as gemfibrozil and fenofibrate, may be used. Fish oil supplements may also be added to the treatment. Many people will need supplementation of 2 to 4 gm fish oil per day to reach goal. 4 gm omega fatty acid per day will reduce triglyceride levels by 30% (2). However, this dosage of fish oil will elevate the LDL by 5-10% and have minimal effect on increasing the HDL. As the capsule dissolves in the stomach, many people also experience a fishy burp which may be minimized or eliminated by freezing the capsules, taking enteric-coated capsules or taking them with food or at bed-time (2). If the triglyceride level is not lowered to < 200 mg/dL with nutrition therapy and treatment of other underlying diseases such as diabetes and hypothyroidism, medical therapy should be initiated.

If the LDL is also elevated, HMG-CoA reductase inhibitors (statins) should be used to lower the LDL to goal based on NCEP ATP III guidelines. Combination therapy for mixed dyslipidemias should be used with caution. All statins, especially at high doses, increase the risk of rhabdomyolysis. Gemfibrozil, when combined with statins, may inhibit statin metabolism and increase serum statin levels. Although fenofibrate/statin therapy has a lower incidence of rhabdomyolysis than gemfibrozil/statin therapy, long-term safety and outcome data for fibrate/statin combination are lacking (4).

Medical and Nutritional Considerations/Treatment for Acute Pancreatitis

The pancreas is a large digestive gland. It is divided into four portions including the head, neck, body and tail (Appendix A, 6). The pancreas, located behind the stomach and opposite the duodenum and the spleen, is the only organ to have both exocrine functions and endocrine functions. The pancreas is composed of two major types of tissues: 1) the acini, ducted exocrine

tissues which are responsible for secreting digestive juices into the duodenum; and 2) the islets of Langerhans, ductless endocrine tissues which secrete the hormones insulin and glucagon into the blood. (6). There are three major types of endocrine cells: alpha cells which secrete glucagon; beta cells are responsible for synthesis and secretion of insulin; and delta cells which secrete somatostatin. Endocrine secretions are carried to the digestive tract via the pancreatic duct which joins with the common bile duct and empties into the duodenum.

Pancreatitis is an inflammation of the pancreas. It is classified as acute (mild or severe) or chronic. Acute pancreatitis is an immunoinflammatory response to a localized process of autodigestion of the pancreatic gland. The trigger event for this response leads to a release of cytokines, inflammatory mediators, and inflammatory cell recruitment with an onset of pain usually within 24-36 hours after the peak of cytokine production. Chronic pancreatitis is a chronic persistent inflammatory state resulting in progressive, irreversible fibrosis and pancreatic insufficiency. The two most common causes of pancreatitis are alcoholism and cholelithiasis. Other risk factors can include medications, hypercalcemia, hyperparathyroidism, infections, pancreatic tumors, autoimmune diseases such as systemic lupus and hypertriglyceridemia. The risk for developing pancreatitis from hypertriglyceridemia becomes clinically significant at levels $>1,000$ mg/dL.

In pregnancy, the incidence of pancreatitis occurs more commonly in the third trimester and postpartum period (7). Hyperlipidemia is the second most common cause of pancreatitis. The total serum triglyceride level during pregnancy is usually less than 300 mg/dL. Cholesterol levels increase and triglyceride levels increase threefold due to high concentrations of circulating estrogen. The usual level of triglycerides required to induce acute pancreatitis is between 750 and 1,000 mg/dL. Symptoms of pancreatitis may include persistent upper abdominal pain

(epigastric and periumbilical) which radiates to the back, chest, flanks and lower abdomen and is generally worse with consumption of food, a low-grade fever, nausea, and vomiting. MJ presented with classic symptoms of radiating abdominal pain, nausea and vomiting. Symptoms of severe acute pancreatitis may also include hypotension, severe abdominal tenderness, abdominal distension, guarding, steatorrhea, and respiratory distress.

There is no single laboratory or clinical sign diagnostic for acute pancreatitis. Amylase and lipase are the most common laboratory markers used to diagnose acute pancreatitis. However, elevated amylase and lipase levels can be non-specific based on the time since onset of pain, other intra-abdominal processes and the presence of other chronic diseases such as renal insufficiency. Plasma lipase is more sensitive and specific than plasma amylase. Other diagnostic markers can include elevated white blood cell count (WBC) and liver function tests. In MJ, a diagnosis of acute pancreatitis was made based on elevated amylase, lipase, white blood cell count (WBC) and liver function studies. Disease severity is determined by the level of cytokines produced, the presence and extent of necrosis, the presence of obesity, infection, failure of at least one organ system and the route of nutrition support. Objective scoring systems such as Ranson criteria and the Acute Physiology and Chronic Health Evaluation (APACHE II) score, and the presence of necrosis on CT scan may identify the severity of acute pancreatitis (Appendix C).

Radiological imaging is used to confirm or exclude the diagnosis, determine the cause, assess severity, detect complications and provide guidance for therapy. Imaging modalities include plain film radiography, abdominal ultrasonography, CT scans, endoscopic retrograde cholangiopancreatography (ERCP), endoscopic ultrasonography, and magnetic resonance cholangiopancreatography (MRCP). Contrast-enhanced CT scan is the standard imaging

technique for detection of pancreatitis. CT scan is not recommended during pregnancy due to the fetal risks from the contrast. Urgent ERCP is indicated in patients with or at risk of biliary sepsis or obstruction. MRCP, a noninvasive technique, has been found to be as accurate as contrast-enhanced CT in predicting severity of pancreatitis. The goal of nutritional management for acute pancreatitis is to minimize pancreatic secretions and to provide adequate protein and energy to meet needs. Nutritional concerns include a rapid deterioration in nutritional status due to increased energy expenditure and increased muscle catabolism and proteolysis; reduced oral intake as a result of abdominal pain, food aversion, nausea, vomiting, continued alcohol abuse or partial obstruction of the duodenum from enlargement of the pancreatic gland; increased nutrient losses due to maldigestion from reduced enzyme output, malabsorption of nutrients, or excessive protein loss from diarrhea or pancreatic fistulas.

Errors in carbohydrate metabolism lead to hyperglycemia and insulin resistance due to increased release of cortisol and catecholamines, increase in glucagon/insulin ratio, impaired beta cell function, and insulin resistance. MJ's did not develop hyperglycemia during her TPN therapy. Electrolyte and micronutrient deficiencies such as hypocalcemia are common. Long-term alcohol abuse can lead to decreases in zinc, magnesium, thiamin, and folate levels. Nutritional intervention for acute pancreatitis should begin as possible to decrease the effects of nutritional concerns. Nutrition support for MJ was initiated after advancement to clear liquid diet failed and lead to exacerbation of her symptoms.

Previously, pancreatic rest (no oral intake and intravenous hydration) was recommended as the sole management strategy to treat pancreatitis by reducing pancreatic stimulation. It was thought that early use of the gut and advancement to oral diet would increase the risk for late complications (8,9). However, that strategy does not appear to affect patient outcome and may

now be considered ineffective. The goal of nutrition therapy is to minimize pancreatic stimulation. Currently, the options for nutrition support in patients with acute pancreatitis include total enteral nutrition (EN), total parental nutrition (TPN), or the use of standard therapy. The type of nutrition support chosen depends on severity of the disease, duration and tolerance. The diet is advanced to liquids and to solids (increased protein and low fat) as the symptoms subside and labs improve. A multivitamin-mineral supplement is also recommended to prevent nutrient deficiencies. Studies have shown that education on food choices is very effective in the treatment of severe acute pancreatitis (9).

Studies have not demonstrated the benefit of nutrition support in mild acute pancreatitis. EN is the first choice over TPN for severe acute pancreatitis if the patient would not be able to tolerate oral feedings within 5-7 days (5). The enteral tube should be placed into the distal portion of the duodenum or into the jejunum. EN has clear benefits in patients with severe acute pancreatitis such as cost-effectiveness, maintenance of gut integrity, increase in antioxidant capacity, faster decrease in C-reactive protein levels, and faster resolution of SIRS compared to patients on TPN. Two meta-analyses showed that use of EN reduces infection by as much as 52%, hospital length of stay by as much as 4 days, surgical intervention by as much as 52% and a trend toward reduced organ failure by as much as 41% when compared to use of PN (9). A meta-analysis concluded that total enteral nutrition is equal to if not better than total parental nutrition (2). More research is needed to determine which type of total enteral nutrition has the most positive effect on patient outcomes. Tolerance to EN can usually be improved with changes in content or infusion rate of the formula.

TPN should be used in patients with severe disease where EN is poorly tolerated (increase in enzyme output, exacerbation of symptoms or disease) or not indicated. In MJ, TPN was initiated

instead of EN based on the anticipated long-term need for nutrition support, development of an ileus, inability to tolerate diet advancement, and comfort of the clinician with TPN versus EN. Lipid emulsion can be included in the TPN formula if the triglyceride level is less than 400 mg/dL and if hypertriglyceridemia is not the cause of pancreatitis (10). Therefore, lipids were initially excluded from the TPN for MJ. If lipids are included in the TPN, a serum triglyceride level should be obtained prior to initiation of TPN. Despite a trial of lipids and administration of Propofol, a lipid based sedative, MJ's triglyceride levels continued to decrease during the hospitalization. At AAMC, TPN orders are written by the PharmD and the RD comments on nutritional adequacy of the TPN formula. It was difficult to get the TPN to adequately meet MJ's caloric needs because of the exclusion of lipids in the TPN formula. Complications of TPN can include catheter occlusion or infection, intestinal atrophy, electrolyte disturbances, hyperglycemia and hypertriglyceridemia. MJ developed an infection to her PICC line which was treated with intravenous antibiotics.

The goal of medical management for acute pancreatitis is to minimize pancreatic secretions, minimize pain and reduce the risk for recurring attacks. Medical management of acute pancreatitis includes aggressive rehydration, maintaining the patient NPO during attacks, pain relief, nutrition support, and medications including antacids (H₂-receptor antagonists or proton pump inhibitors) to reduce gastric acid secretion and maintain optimal intestinal pH. Monitoring of hemodynamic status and laboratory/serum parameters for complications is also important (6).

There is very limited information on the use of complementary and alternative (CAM) therapies in hypertriglyceridemia and pancreatitis. Grape seed extract is used for high cholesterol. Preliminary clinical trials have shown that grape seed extract has some beneficial antioxidant effects (11). However, there is little scientific evidence for specific conditions. CAM

therapies that may be used for hypertriglyceridemia include guggul (an Ayurvedic therapy), fenugreek, psyllium seed, red yeast rice. Grape seed extract and antioxidants such as vitamin C, vitamin E, betacarotene, and selenium have been mentioned for prevention and treatment of pancreatitis (12,13). However, there is no scientific evidence that these therapies actually work.

Implications of findings for practice of dietetics include:

- Family history can be very valuable in assessing a patient's risk for disease and in providing appropriate nutritional therapy.
- Application of the Nutrition Care Process: The steps of assessment, goal, diagnosis, intervention, monitoring and evaluation are applicable for effective nutritional management of hypertriglyceridemia and pancreatitis.
 - **Goal:** Pt will be able to identify 3 low-fat food choices and 3 high fiber food choices by reading food labels.
 - **Diagnosis:** Food and Nutrition-related knowledge deficit related to low-fat, high fiber food choices as evidenced by frequent consumption of ice cream and simple carbohydrates.
 - **Intervention:** Instruct patient on reading labels for identification of fat and fiber content. Instruct on low-fat and high fiber food choices.
 - **Monitoring and Evaluation:** Lab values after 3-6 months, food recall, patient response to questions about food sources of fat and fiber.
- Nutrition education has been shown to be effective in the management of hypertriglyceridemia and pancreatitis.
- TPN was previously considered the gold standard for nutrition therapy in patients with severe acute pancreatitis. However, enteral nutrition is now the preferred method

of nutrition support. Studies have shown that EN is equal to if not better than TPN when the tube is placed in the distal portion of the duodenum or the jejunum.

Tolerance issues with EN can usually be resolved with modification of TPN composition or adjustment in infusion rate. Although EN is recommended, TPN continues to be used more often than EN due to the comfort of the clinician and concern regarding pancreatic stimulation with EN.

- TPN orders written by PharmD versus an RD: If TPN orders are written by a PharmD, an RD is an essential member of the team for assessment of nutritional adequacy of nutrition support and to provide recommendations to the PharmD.

APPENDICES

Appendix A: Figures (14,15)

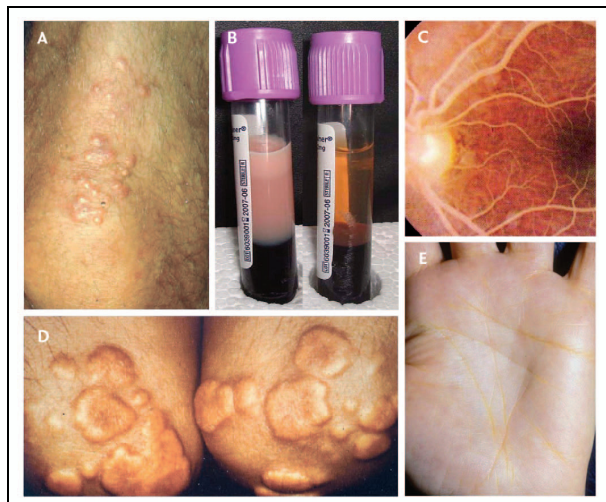


Fig. 1. Clinical manifestations of primary hypertriglyceridemia. A: Eruptive cutaneous xanthomas. B: Lipemic plasma. Whole blood has been allowed to stand at 4°C overnight. The sample on the right comes from a normolipidemic subject. C: Lipemia retinalis. D: Tuberous xanthomas. E: Palmar crease xanthomas.

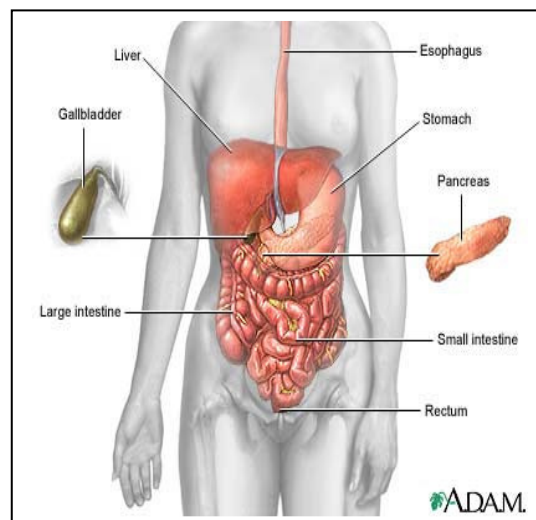


Figure 2. The esophagus, stomach, large and small intestine, aided by the liver, gallbladder and pancreas convert the nutritive components of food into energy and break down the non-nutritive components into waste to be excreted (<http://www.drugs.com/enc/pancreatitis.html>).

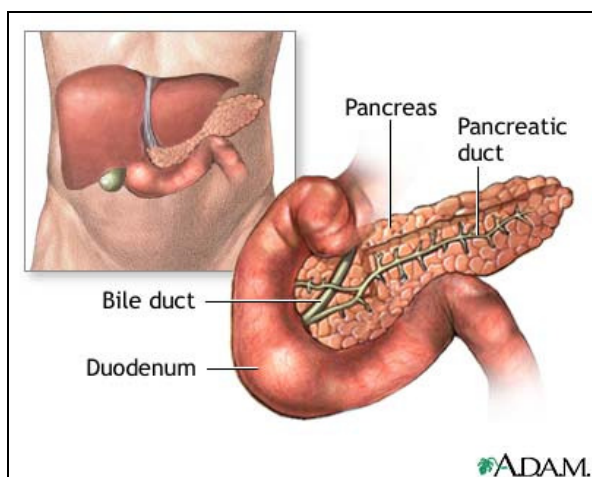


Figure 3. The pancreas is located posterior to the abdomen. It contains cells that secrete the hormone insulin, and cells that secrete digestive enzymes that aid in the breakdown of food in the gastrointestinal tract. The pancreas secretes these enzymes into the pancreatic duct, which joins the common bile duct from the liver and drains into the small intestine (<http://www.drugs.com/enc/pancreatitis.html>).

Appendix B: Pharmacological Therapies for Hypertriglyceridemia (4)

Therapy	TG reduction (%)	LDL increase/reduction (%)	HDL increase (%)	Possible side effects Pregnancy/Lactation Concerns
Statins- Lipitor, Lescol, Mevacor, Pravachol, Crestor, Simvastin	20-40	18-55 ↓	5-15	Elevated liver enzymes Pregnancy category C
Fenofibrates Tricor, Lopid	40-60	5-30 ↑	15-25	Rhabdomyolysis, especially with gemfibrozil/statin combination Pregnancy Category C
Niacin OTC immediate release, OTC sustained release, Prescription	30-50	5-25 ↓	20-30	Flushing, elevated liver enzymes Pregnancy Category C
Fish oil OTC omega- 3-acid capsules Omacor	30-50	5-10 ↑	5-10	Fishy aftertaste, GI upset. Pregnancy Category C
TG= triglyceride; LDL-C= low-density lipoprotein cholesterol; HDL-C= high-density lipoprotein cholesterol; OTC= over the counter; EPA= eicosapentaenoic acid; DHA=docosahexaenoic acid.				

Appendix C: Ranson criteria: Signs for classification of severity of acute pancreatitis (10)**At time of admission with diagnosis:**

- Age >55 years
- White blood cell count >16,000/mm
- Blood glucose >200 mg/dL
- Serum lactate dehydrogenase (more than twice normal)
- Serum glutamic-oxaloacetic transaminase (more than six times normal)

After initial 48 hours:

- Decrease in hematocrit of >10%
- Serum calcium <8 mg/dL
- Increase in blood urea nitrogen of >5 mg/dL
- Arterial pO₂ <60 mmHg
- Base deficit >4 mEq/L
- Estimated fluid sequestration 6,000 mL

Appendix D: FDA Categories of Drug Safety During Pregnancy (16)

Category	Description
A	Controlled human studies show no fetal risks; these drugs are the safest.
B	Animal studies show no risk to the fetus and no controlled human studies have been conducted, or animal studies show a risk to the fetus but well-controlled human studies do not.
C	No adequate animal or human studies have been conducted, or adverse fetal effects have been shown in animals but no human data are available.
D	Evidence of human fetal risk exists, but benefits may outweigh risks in certain situations (eg, life-threatening disorders, serious disorders for which safer drugs cannot be used or are ineffective).
X	Proven fetal risks outweigh any possible benefit.

Appendix E: Medications (16)

Medication	Indication	Food/Drug Interaction
Fentanyl patch	Analgesic, Narcotic	↑ Amylase and Lipase Anorexia
Propofol	Sedative	↓ Renal function ↑Triglycerides Not with egg or soy allergy
Levaquin	Antibiotic	Orange juice ↓ drug level 2 hours before or after antacids
Furosemide	Diuretic	Avoid natural licorice ↓ K, Mg, Na
Novolog SSI	Hypoglycemic	↑ Weight Alcohol ↑ hypoglycemia
Enoxaparin	Anticoagulant	Not with pork allergy- from pork intestinal mucosa
Promethazine HCL	Antiemetic	↑ Glucose Avoid Alcohol
Metronidazole	Antibiotic, Antimicrobial	Metallic taste Avoid alcohol, may cause disulfuram-like reaction
Labetalol	Antihypertensive	Avoid natural licorice May mask symptoms hypoglycemia
Acetaminophen	Analgesic, Antipyretic	↑ Vitamin C ↑toxicity ↑ Alcohol ↑hepatotoxiicty

Appendix F: Labs

Lab	Normal Value	9/16	9/24	9/30	10/4	11/5*
Na	136-144 mEq/L	127	130	136	137	142
K	3.5-5.0 mEq/L	5.6	4.4	4.2	4.2	3.9
BUN	8-23 mg/dL	<5	13	14	10	<5
Crea	0.4-1.2 mg/dL	0.6	0.6	0.8	0.7	0.9
Glu	70-99 mg/dL	110	89	99	85	107
WBC	5-10 x 10 ³ /mm ³	15.3	19.7	12.9	13.9	4.5
Platelets	177- 406 x 1000/ μ L	161	222	499	500	191
Mg	1.3-2.1 mEq/L	-----	2.2	2.0	1.9	-----
Phos	2.3-4.3 mg/dL	-----	3.4	3.9	4.2	4.4
Chol	Desirable 120-199 mg/dL	514	122	122	101	149
TG	Desirable < 150 mg/dL	726	398	264	381	291
HDL	Desirable 40-60 mg/dL	17	<17	----	-----	----
LDL	Optimal < 100 mg/dL	29	38	-----	-----	-----
Amylase	30-110 U/L	123	83	141	138	113
Lipase	16-63 U/L	448	51	95	94	77
LDH	105-230 U/L	-----	425	420	487	167
AST	10-31 U/L	38	40	37	42	38
ALT	4-31 U/L	8	15	16	27	22
*2 nd Admission.						

Appendix G: TPN Prescription

Nutrient	9/24	9/27	9/30	10/1	10/4
AA	60	60	60	60	50
Dextrose	375	275	250	250	250
Lipid	None	None	35	35	15
Protein	60	60	60	60	50
Total calories (kcal/kg)	1175 (18)	1175 (18)	1505 (23)	1505 (23)	1200 (18)
Calorie requirement kcal/kg	1800 (28)	1800 (28)	1800 (28)	1800 (28)	1800 (28)
% Calorie requirement	65	65	85	85	75
Protein requirement gm/kg	60 (0.9)	60 (0.9)	60 (0.9)	60 (0.9)	60 (0.9)
% Protein requirement	100	100	100	100	83

GLOSSARY OF TERMS

Acute pancreatitis: Sudden inflammation of the pancreas. The pancreas abruptly becomes inflamed and then gets better. Attacks may reoccur after full recovery.

Apolipoproteins: Proteins associated with lipids that assist with their assembly, transport, and metabolism.

Computerized axial tomography (CAT scan): Pictures of structures within the body created by a computer.

Chronic pancreatitis: A form of pancreatitis in which there is persistent inflammation of the pancreas.

Endocrine: Cells or tissue that produce hormones released into the bloodstream and that have an effect on other cells.

Endoscopic retrograde cholangiopancreatography: ERCP is a procedure that combines x-rays and an endoscope (a long, flexible, lighted tube) to diagnose and treat problems in the liver, gallbladder, bile ducts, and pancreas, including gallstones, inflammatory strictures (scars), leaks (from trauma and surgery), and cancer by injecting dye into the bile ducts and pancreas. Possible complications of ERCP include pancreatitis (inflammation of the pancreas), infection, bleeding, and perforation of the duodenum.

Eruptive xanthomas: 1- to 3-mm yellow papules that can erupt anywhere but are usually seen on the back, chest, and proximal extremities.

Exocrine: Cells or tissue that produce substances that are released through a duct and into another organ.

Lipemia retinalis: Visualization of lipemic blood in the retinal blood vessels.

Metabolic syndrome: Metabolic syndrome includes a group of complications such as abdominal obesity, insulin resistance, low high-density lipoprotein (HDL), high triglyceride, and hypertension which increase the risk for cardiovascular disease.

Magnetic resonance cholangiopancreatography (MRCP): A non-invasive procedure which uses magnetic resonance imaging to visualize the biliary and pancreatic ducts.

Palmar xanthomas: yellow creases on the palm which are typically seen in type III hyperlipidemia.

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