PANCREATITIS
NORMAL PANCREATIC FUNCTION
Pancreas

- Elongated flattened gland that lies in the upper abdomen behind the stomach
- Endocrine and exocrine functions
Islets of Langerhans

Four major types of cells
- Beta cells: insulin & amylin
- Alpha cells: glucagon
- Pancreatic polypeptide cells: Pancreatic polypeptide
- Delta cells: somatostatin
Exocrine

- Functional unit is an acinus and its draining duct
- All the ducts come together to form the main pancreatic duct
- Acinar cells produce, store, and secrete digestive enzymes
- Zymogen granules
Ducts

- Individual draining ducts
- Main pancreatic duct
- Merging with Common Bile Duct
- Ampulla of Vater
Secretions

- Secretes bicarbonate rich fluid
- Most enzymes secreted in inactive form
  - Activated in lumen of intestine
- Enzymes
  - Amylase
  - Lipase
  - Proteases
- Regulated by humoral and neural responses
Pancreatic Secretions during Meal

- **Cepalic phase**
  - Initiator: Vagus nerve, sight, smell, and taste of food
  - secretion of bicarbonate and pancreatic enzymes

- **Gastric phase**
  - Initiator: Gastric distension
  - Enzyme rich, low volume secretion

- **Intestinal phase**
  - Initiator: Cholecystokinin and Secretin
  - High volume secretions
Stellate Cells

- Role in secretion and modulation of extracellular matrix
- When activated assume a stellate or myofibroblastic appearance
- Also stimulated by inflammatory cytokines released in acinar cell necrosis
- Activated cells found in areas of extensive necrosis and inflammation
ALCOHOL
Standard Drink

- 15g
  - 12 oz beer
  - 10 oz wine cooler
  - 5 oz wine
  - 1.5 oz hard liquor
- Takes the average person about 2 hours to completely metabolize
Absorption

- Rapidly absorbed
- Absorbed in the duodenum and jejunum
- Readily dispersed throughout the body
Metabolism

- Low-moderate intakes
  - Alcohol dehydrogenase (ADH) pathway

\[ \text{Ethanol} \rightarrow \text{Acetaldehyde} \rightarrow \text{Acetic acid} \]
Metabolism

- Moderate-excessive intakes
  - Microsomal ethanol oxidizing system (MEOS)
    - Requires energy to operate
    - Potential for drug toxicities
  - Catalase pathway
    - Minor contribution
## Metabolism

<table>
<thead>
<tr>
<th>Alcohol Metabolic Pathway</th>
<th>Main Location of Pathway Activity</th>
<th>Alcohol Intake Level That Activates Pathway</th>
<th>Extent of Participation in Alcohol Metabolism</th>
</tr>
</thead>
<tbody>
<tr>
<td>ADH pathway</td>
<td>Stomach Liver (mostly)</td>
<td>Low to moderate intake</td>
<td>Major role (metabolizes about 90% of alcohol)</td>
</tr>
<tr>
<td>MEOS</td>
<td>Liver</td>
<td>Moderate to excessive intake</td>
<td>Role increases in importance with increasing alcohol intake levels</td>
</tr>
<tr>
<td>Catalase pathway</td>
<td>Liver Other cells</td>
<td>Moderate to excessive intake</td>
<td>Minor</td>
</tr>
</tbody>
</table>
Pancreatic Consequences

- Decreases pancreatic lipase secretion
  - Poor absorption of fat and fat-soluble vitamins
- Impairs normal function
  - Related hypoglycemia
- Increases risk of pancreatic cancer
Acute vs. Chronic Pancreatitis

**ACUTE:**
- Acute inflammatory process of the pancreas with variable involvement of other regional tissues or remote organ systems
- Sudden swelling and inflammation of the pancreas
- Complete recovery of pancreas after episode

**CHRONIC:**
- Permanent and irreversible damage of the pancreas, with evidence of chronic inflammation, fibrosis, and destruction of exocrine and endocrine tissue
Acute: Pathophysiology

- Initiated:
  - by injury to acinar cells or impairment of enzymes secretion

- Leads to local inflammatory complications, a systemic response, and sepsis:
  - Microcirculatory changes
  - Vascular permeability and resulting edema
  - Reperfusion of damaged pancreatic tissue
  - Activation of complement and release of C5a
  - Macrophages recruitment
  - SIRS
Sepsis: Defining a Disease Continuum

- **Infection/Trauma**
- **SIRS**
- **Sepsis**
- **Severe Sepsis**

- A clinical response arising from a nonspecific insult, including ≥2 of the following:
  - Temperature ≥38°C or ≤36°C
  - HR ≥90 beats/min
  - Respirations ≥20/min
  - WBC count ≥12,000/mm³ or ≤4,000/mm³ or >10% immature neutrophils

- SIRS with a presumed or confirmed infectious process

- Sepsis with ≥1 sign of organ failure
  - Cardiovascular (refractory hypotension)
  - Renal
  - Respiratory
  - Hepatic
  - Hematologic
  - CNS
  - Metabolic acidosis

**SIRS** = systemic inflammatory response syndrome.
Severity

- Classified as mild or severe acute pancreatitis
- Mild:
  - interstitial pancreatitis
  - Minimal to no extrapancreatic organ dysfunction
- Severe:
  - Organ failure
  - Local complications: necrosis, abscess, pseudocyst
Two stages of Acute Pancreatitis

1) Inflammatory Cascade
   - Systemic inflammatory response
   - Evolves dynamically with variable degrees of pancreatic and peripancreatic ischemia or edema
   - Evolves either to resolution or irreversible necrosis, liquefaction and development of fluid collections in and around the pancreas
   - 75-85% of patients have resolution
   - Lasts one week
Two stages of Acute Pancreatitis

2) Necrotizing process

- Pancreatic and peripancreatic fat necrosis
- Acute Fluid collection
- Pseudocyst
- Abcess
- WOPN
- Organ failure
- Lasts weeks to months
Chronic: Pathophysiology

- In affected lobules, acinar cells are surrounded and replaced by fibrosis
- Infiltration of fibrotic area with lymphocytes and macrophages
- Fibrosis progresses within lobules and between lobules becoming more widespread
- Pancreatic ducts abnormal with progressive fibrosis: stricture formation and dilation
- Ductal protein plugs form
Fibrosis

- Replacement of normal cells with fibrous tissue
- Sign that interstitial stellate cells are activated
SIGNS AND SYMPTOMS
Acute: Signs and Symptoms

- Abdominal pain, tenderness
- Fever, N/V, sweating
- Clay-colored stools
- Gaseous abdominal fullness
- Edema
- Indigestion
- Yellowing of skin and whites of eyes (Jaundice)
- Skin rash or sore (lesion)
- Swollen abdomen
Chronic: Signs and Symptoms

- Abdominal pain
- Diarrhea, nausea, vomiting
- Steatorrhea
- Pale or clay colored stools
- Chronic weight loss
- Diabetes Mellitus
ETIOLOGY
Acute Pancreatitis: Etiology

- Obstruction
  - Gallstones, Tumor
- Chronic Alcohol Abuse
- Medications
- Metabolic
- Infections
- Trauma
- Post-ERCP
Obstruction
Gallstones

- Causes 40% of cases but only 3-7% with gallstones get acute pancreatitis
- More common in women
- Obstruction
- Small stones <5mm cause ampullary obstruction
- Cholecystectomy and clearing bile duct of stones will prevent recurrence
Causes: Medications

- Infrequent but important cause
- >120 drugs implicated to cause Acute Pancreatitis
- Rechallenge for evidence

Mechanisms

1) hypersensitivity reaction
2) accumulation of toxic metabolite
3) overdose of drugs with intrinsic toxicity
Causes: Medications

Table 58-4 Drugs Associated with Acute Pancreatitis*

<table>
<thead>
<tr>
<th>Acetaminophen</th>
<th>Hydrocortisone</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alphamethyldopa</td>
<td>Ifosfamide</td>
</tr>
<tr>
<td>5-Aminosalicylic acid compounds</td>
<td>Interferon-α</td>
</tr>
<tr>
<td>Sulfasalazine</td>
<td>Isoniazid</td>
</tr>
<tr>
<td>Azodisalicylate</td>
<td>Lamivudine</td>
</tr>
<tr>
<td>Mesalamine</td>
<td>Lisinopril</td>
</tr>
<tr>
<td>L-Asparaginase</td>
<td>Losartan</td>
</tr>
<tr>
<td>Azathioprine</td>
<td>Meglumine</td>
</tr>
<tr>
<td>Benazepril</td>
<td>6-Mercaptopurine</td>
</tr>
<tr>
<td>Bezafibrate</td>
<td>Methimazole</td>
</tr>
<tr>
<td>Cannabis</td>
<td>Metronidazole</td>
</tr>
<tr>
<td>Captopril</td>
<td>Nelfinavir</td>
</tr>
<tr>
<td>Carbazochrome</td>
<td>Norethindrone/mestrol</td>
</tr>
<tr>
<td>Cimetidine</td>
<td>Pentamidine</td>
</tr>
<tr>
<td>Clozapine</td>
<td>Pravastatin</td>
</tr>
<tr>
<td>Codeine</td>
<td>Procaainamide</td>
</tr>
<tr>
<td>Cytosine arabinoside</td>
<td>Pyritinol</td>
</tr>
<tr>
<td>Dapsone / Didanosine</td>
<td>Simvastatin</td>
</tr>
<tr>
<td>Dexamethasone</td>
<td>Sulfamethazole</td>
</tr>
<tr>
<td>Enalapril</td>
<td>Sulfamethoxazole</td>
</tr>
<tr>
<td>Erythromycin</td>
<td>Stibogluconate</td>
</tr>
<tr>
<td>Estrogen</td>
<td>Sulindac</td>
</tr>
<tr>
<td>Fluvasatin</td>
<td>Tetracycline</td>
</tr>
<tr>
<td>Furosemide</td>
<td>Trimethoprim-sulfamethoxazole</td>
</tr>
<tr>
<td>Hydrochlorothiazide</td>
<td>Valproic acid</td>
</tr>
</tbody>
</table>

*Class 1 and class 2 drugs. For class 1 drugs: two or more case reports published, absence of other causes of acute pancreatitis, rechallenge documented in at least one report. For class 2 drugs: four or more case reports published, absence of other causes of acute pancreatitis, consistent latency in at least 75% of cases published.

Causes: Metabolic

- Hypertriglyceridema
  - 3rd most common cause
  - Serum triglycerides >1000mg/dL
  - Lactescent (milky) serum
  - Mechanism unclear

- Hypercalcemia
  - Rare
Causes: Infections

- Infectious agents cause inflammation of pancreas
- Determine this is the cause by finding infectious agent in pancreas or pancreatic duct
- Also characteristic symptoms of infectious agent occurring at same time as pancreatitis symptoms
- Viruses, MMR Vaccine, Bacteria, fungi
  - Mumps, Herpes Simplex virus, salmonella, tuberculosis
Causes: Trauma

- Penetrating trauma or blunt trauma
  - Blunt: compression of pancreas by spine

- Trauma can range from mild contusion to severe crush injury or transection of the gland

- Damage to acinar cells
Causes: Post-ERCP

- Endoscopic retrograde cholangiopancreatography
- ERCP is a diagnostic procedure to examine diseases of the liver, bile ducts and pancreas
- Use a duodenoscopy to view inside structures
- Pancreatitis is the most common complication
  - Irritation of the pancreas
Chronic Pancreatitis: Etiology

- Alcohol
- Genetic
- Autoimmune pancreatitis
- Obstructive
- Recurrent of Severe Acute Pancreatitis
- Idiopathic
Causes: Alcoholism

- Most common
- Alcohol & its metabolites have direct injurious effects on pancreatic acinar cells
- Increases acinar cell sensitivity to physiologic stimuli
- Promotes inflammatory responses
- Injury to ductal cells
- Stimulates pancreatic stellate cells
- Form ductal injury and ductal stones
Causes: Alcoholism
Causes: Genetic

- Mutations in the PRSS1, SPINK1, or CFTR
- Increases susceptibility or pace and severity
- Usually a combination
Autoimmune Pancreatitis

- Dense infiltration of pancreas and other organs by lymphocytes and plasma cells
- Express IgG4
- Target unknown
Incidence and Prevalence

ACUTE PANCREATITIS

- 4.8 to 38 cases per 100,000
- 100,000 hospitalizations
- 2,000 die per year from associated complications
- 14th most common cause of GI related deaths
- Cost $2.5 billion in 2000

CHRONIC PANCREATITIS

- 4 cases per 100,000
- 56,000 hospitalizations per year
- 122,000 outpatient visits per year
RISK FACTORS
Risk Factors

- High blood triglyceride levels
- Hyperparathyroidism
- Cystic fibrosis
- Blockage of pancreatic duct
- Autoimmune complications
- Alcohol abuse
- Smoking
- Injury to pancreas from an accident
- More common in men
- Ages 30-40 yrs
Co-morbidities

- Multiple organ failure
- Tetany
- Diabetes Mellitus
- ARDS
- SIRS
- Calcification of pancreas
- Ascites
Pancreatitis and DM

- Most common in chronic
- Tissue and cells are destroyed
  - Beta cells produce insulin
DIAGNOSIS
Diagnosis: Acute

Suspected from clinical features
Confirmed by labs and imaging tests
  - Serum Amylase: 3x UL
  - Serum Lipase: 3x UL
  - Ranson’s Criteria evaluation (see next slide)
  - CT scans
Ranson Criteria and CT evaluation scores predict outcomes.

<table>
<thead>
<tr>
<th>Variables of the Ranson Criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Ranson Criteria</strong></td>
</tr>
<tr>
<td><strong>For Acute Non-Gallstone Pancreatitis</strong></td>
</tr>
<tr>
<td><strong>Upon admission:</strong></td>
</tr>
<tr>
<td>1. Age</td>
</tr>
<tr>
<td>2. WBC</td>
</tr>
<tr>
<td>3. Glucose</td>
</tr>
<tr>
<td>4. LDH</td>
</tr>
<tr>
<td>5. AST</td>
</tr>
<tr>
<td><strong>Within 48 hours:</strong></td>
</tr>
<tr>
<td>1. Drop in HCT</td>
</tr>
<tr>
<td>2. Serum Ca</td>
</tr>
<tr>
<td>3. Base deficit</td>
</tr>
<tr>
<td>4. Increase BUN</td>
</tr>
<tr>
<td>5. Fluid deficit</td>
</tr>
<tr>
<td>6. Arterial PO₂</td>
</tr>
</tbody>
</table>
Mild vs Severe Acute Pancreatitis

Severe Acute associated with:

- Organ failure
- Local complications:
  - necrosis
  - Abscess
  - pseudocyst
  - Peptic ulcer
  - Ischemia
  - Bowel obstruction
  - Choleystitis (inflammation of gallbladder)
- High score from Ranson’s criteria

Diagnosis based on detection of systemic and/or local complications
Diagnosis: Chronic

Recurrent episodes of acute Labs

Chronic abdominal pain- some patients may not have pain or experience spontaneous remission of pain by organ failure- pancreatic burnout theory

Clinical presentations (ABC’s)

Steatorrhea       malabsorption
Vitamin deficiencies Diabetes
Weight loss

Positive diagnostic tests and CT tests
Diagnosis: Chronic (Imaging Studies)

- CT scan
- Abdominal ultrasound
Magnetic resonance cholangiopancreatography

ERCP
TREATMENT
Treatment: Acute Mild

- Fluids
- Analgesia
- Nutrition
- Nasogastric Suction
- Acid suppression
- Somatostatin/ Octreotide
Treatment: Acute Severe

- Aggressive fluid resuscitation
- Oxygen
- Pain relief
- Nutrition
Treatment: Chronic

- Treatment Goals
  - Relieve acute/chronic pain
  - Calm disease to prevent recurrent attacks
  - Treat/correct diabetes and malnutrition
  - Manage complications
Nutritional/ Medical Support: Chronic

- Cessation of alcohol and tobacco use
- Analgesics
- Decompression
- $H_2$ receptor antagonist/ proton pump inhibitor
- Somatostatin/ Octreotide
- Antioxidants
- Vitamin Supplementation (A, D, E, K, and B12)
- Low-fat diet and small meals
- Pancreatic enzyme preparations
Prognosis: Acute

- Most cases go away in about a week
- Can develop into a life-threatening illness
- Pancreatitis can return- likelihood depends on the cause and the success of treatment
- Death rate is high If patient experiences: hemorrhagic pancreatitis liver, heart, or kidney impairment Necrotizing pancreatitis
Prognosis: Chronic

- Progressive and irreversible loss of pancreatic structure and exocrine/endocrine function
- Surgery: $\frac{1}{2}$ of all patients will undergo surgery during the course of their disease
- Appropriate when initial medical and endoscopic treatments fail to relieve abdominal pain
- Disability
- Death
MEDICAL NUTRITION THERAPY
### Normal Pancreatic Function

#### Exocrine Function
- Secretion of the enzymes amylase, carboxylesterase, sterol esterase, lipase, Dnase, Rnase, and more
- Aide in digestion of proteins, fats, and carbohydrates

#### Endocrine Function
- Manufactures insulin, glucagon, and somatostatin for absorption into the bloodstream
Medical Nutrition Therapy

- **Acute pancreatitis**
  - NPO for 5-7 days
    - Hydration maintained intravenously
    - Less severe attacks may be on a liquid diet that has minimal fat.
  - If pancreatitis hasn’t resolved itself within 5-7 days, start enteral nutrition.
    - Feeding into jejunum, past the Ligament of Trietz, bypasses cephalic and gastric phases of exocrine pancreatic stimulation.
    - Use a standard formula for these patients, but if case is still not resolved then switch to an elemental formula.
• Severe acute pancreatitis
  • In prolonged acute cases if enteral isn’t being tolerated, PN may be necessary.
  • Patients in severe stress may be experiencing some glucose intolerance. Because of this they will generally require a mixed fuel system of dextrose and lipid to avoid complications.
  • Most patients will need an insulin drip because of endocrine abnormalities.
  • If hypertriglyceridemia is causing the pancreatitis then the PN regimen should not include a lipid emulsion.
  • Only patients with triglycerides levels <400 mg/dl may be given lipids. Use a 3 in 1 solution and monitor TG levels.
  • If TGs are >400mg/dl, use a dextrose-base solution, monitor serum glucose frequently.
MNT continued…

• **Chronic pancreatitis**
  • Oral diet is similar as in acute pancreatitis, but has a few small changes:
    • Needs supplemental pancreatic enzymes. Enteric-coated minimicrospheres are preferred because they are effective in treating steatorrhea, and they protect the enzymes from gastric acids.
    • They need supplemental fat-soluble vitamins, vitamin B12, and bicarbonate.
    • Insulin and diabetes education
# Nutritional Management of Acute vs. Chronic Pancreatitis

## Acute:
- Withhold oral and enteral feeding
- Support with IV fluids
- If oral nutrition cannot be initiated in 5 to 7 days, start nutrition support
- For less severe cases of prolonged acute pancreatitis, TF can be initiated beyond the ligament of Treitz using a polymeric formula
- For severe acute pancreatitis, PN should be initiated
  - If TGs are <400 mg/dl before PN initiation, use a 3-in-1 solution and monitor TG levels
  - If TGs are elevated (>400 mg/dl), use a dextrose-based solution, monitor serum glucose frequently, and treat as needed with insulin
- Once oral nutrition is started, provide
  - Easily digestible foods
  - Low-fat diet
  - 6 small meals
  - Adequate protein intake
  - Increased calories

## Chronic:
- Provide oral diet as in acute phase
- TF can be used when oral diet is inadequate
- Supplement pancreatic enzymes
- Supplement fat-soluble vitamins, vitamin B12, and bicarbonate
PANCREATIC CANCER
Statistics

• Pancreatic cancer is the 4\textsuperscript{th} leading cause of cancer death in men and women.
• The prognosis is poor. Combining all stages of pancreatic cancer, the one-year survival rates are 24% while the five-year survival rate are only 5%.
• Smoking, obesity, and diabetes have all been shown to increase the risk for developing pancreatic cancer.
Pancreatic Cancer

**Signs and Symptoms**

- Most patients lack any signs or symptoms until late in the disease, which delays diagnosis.
- The first signs are often jaundice that results from a tumor obstructing the extrahepatic bile duct.

**Diagnosis**

- The preferred method of diagnosis for pancreatic tumors is a CT scan.
Relationship between Pancreatitis and Pancreatic Cancer?

- In a study of 38,000 chronic pancreatitis patients it was observed that patients with chronic pancreatitis inflammation, had an increased risk of developing pancreatic cancer.

- This risk has been increasingly observed especially as survival rates of CF patients have increased. This should be watched in adolescents and adults with unexplained complaints originating from the abdominal organs.
Whipple Procedure
The Whipple pancreaticoduodenectomy is the most common operation for pancreatic cancer. In the past it has been shown to have high morbidity and mortality rates, but it has been showing a decrease in mortality and complications due to a variety of things such as advancements in surgery, better ICUs, and advances in anesthesia, antibiotics, and interventional radiology. Even with potentially curative surgery prognosis remains poor with a median survival rate of 10.5 to 20 months.
Case Study

- EJ

30 year old Female

Occupation: Pharmaceutical sales

Chief Complaint: bouts of epigastric pain that radiates to back, lasting 4 hours to several days, recent unintentional weight loss

Onset of symptoms symptoms 12 months ago

Alcohol use: since high school, 2-3 alcoholic beverages a night
Anthropometric

Ht: 5’8”
Wt: 112
Wt one year ago: 140
- 20% wt change
BMI: 17.2
%IBW: 81%
### Biochemical

**Abnormal Labs:**

<table>
<thead>
<tr>
<th>Test</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Transferrin</td>
<td>155 (low)</td>
</tr>
<tr>
<td>Glucose</td>
<td>130 (high)</td>
</tr>
<tr>
<td>Bilirubin</td>
<td>1.5 (high)</td>
</tr>
<tr>
<td>AST</td>
<td>50 (high)</td>
</tr>
<tr>
<td>LDH</td>
<td>323 (normal)</td>
</tr>
<tr>
<td>Alk Phosphate</td>
<td>178 (high)</td>
</tr>
<tr>
<td>CPK</td>
<td>245 (high)</td>
</tr>
<tr>
<td>Cholesterol</td>
<td>225 (high)</td>
</tr>
<tr>
<td>HDL</td>
<td>40 (low)</td>
</tr>
<tr>
<td>Triglycerides</td>
<td>250 (high)</td>
</tr>
<tr>
<td>WBC</td>
<td>$14.5 \times 10^3/mm^3$ (high)</td>
</tr>
<tr>
<td>HCT</td>
<td>35.7g/dl (low)</td>
</tr>
<tr>
<td>MCV</td>
<td>101.5 um$^3$ (high)</td>
</tr>
</tbody>
</table>
Clinical

- Thin
- Temporal muscle wasting
- Appears to be in discomfort
- No edema
- Bowl sounds normal
- Tenderness in epigastric religion
- Liver and spleen not enlarged
Dietary Assessment

24 hour recall

Breakfast:
- Dry bagel
- 8 oz of black coffee

Lunch:
- 16 oz Diet Coke
- Lean Cuisine

Dinner:
- 15oz white wine
- 2-3oz Grilled salmon
- Baked Potato with butter and sour cream
- Two stalks of broccoli w/cheese sauce

Total Calories: 1327 kcal
Total Protein: 54 grams
Total Fat: 38 grams
Nutritional Assessment

- Estimated Calorie Needs:
  - Harris Benedict: 1180 kcals
  - Add stress factor of: $1180 \times 1.2 = 1418$ kcal
  - $1428 \text{ kcal} + 300 \text{ kcal} = 1728 \text{ kcal}$

- Protein Needs: $1.0 \text{ g/kg} = 51 \text{ grams}$
PES Statement

Impaired nutrient utilization related to chronic pancreatitis as evidenced by steatorrhea and severe unintentional weight loss of ten pounds in the last month.
Nutritional Intervention

- Add vitamin supplement.
- Pancreatic Enzyme replacement
- Recommend low fat diet with small meals
- Encourage her to stop alcohol consumption
Sample Diet

- **Breakfast:**
  - 1 cup instant oatmeal
  - 1 cup skim milk
  - 1 medium banana

- **AM Snack:**
  - 6oz yogurt with ¼ cup granola

- **Lunch**
  - Turkey Sandwich with Lettuce, Tomato, and low-fat mayo
  - 1 cup skim milk
  - 10 baby carrots

- **PM Snack**
  - 1 cup popcorn
  - 1 medium apple

- **Dinner**
  - 3 oz BBQ chicken breast
  - 1 cup wild rice
  - Green salad with fat-free dressing
  - 1 cup skim milk

- **HS Snack**
  - 1 cup raspberry sherbet

- **Total Calories:** 1700 kcal
- **Total Protein:** 81 grams
- **Total Fat:** 19 grams
References:

- Judd AM. Lecture slides. Pathophysiology, Brigham Young University, September 19, 2011.