THE ANATOMY AND PHYSIOLOGY OF THE GALL BLADDER

The gallbladder is situated on the underside of the liver and the right side of the abdomen. The biliary tract or biliary tree is the anatomical path that bile takes after it is secreted by the liver and concentrated in the gallbladder, and before it arrives in the duodenum. Hence, the main function of the gallbladder is to concentrate, store, and excrete bile, which is produced in the liver. Bile leaves the liver via the common hepatic duct, which merges with the cystic duct from the gallbladder to form a common bile duct. The common bile duct merges with the pancreatic duct, forming the ampulla of Vater, which enters the duodenum.

About 95 percent of the bile salts are reabsorbed into the blood from the small intestine, about in the upper half by diffusion through the mucosa in the upper part of small intestine and half by, active transport in the intestinal mucosa of the distal ileum. This recirculation of bile salts is called enterohepatic circulation. When food reaches the duodenum, the release of intestinal hormones such as cholescystokinin and secrtin is initiated. This stimulates the gallbladder and pancreas and causes relaxation of sphincter of Oddi to relax, allowing pancreatic juice and bile to flow into the duodenum at the ampulla of Vater to assist in fat digestion and absorption.

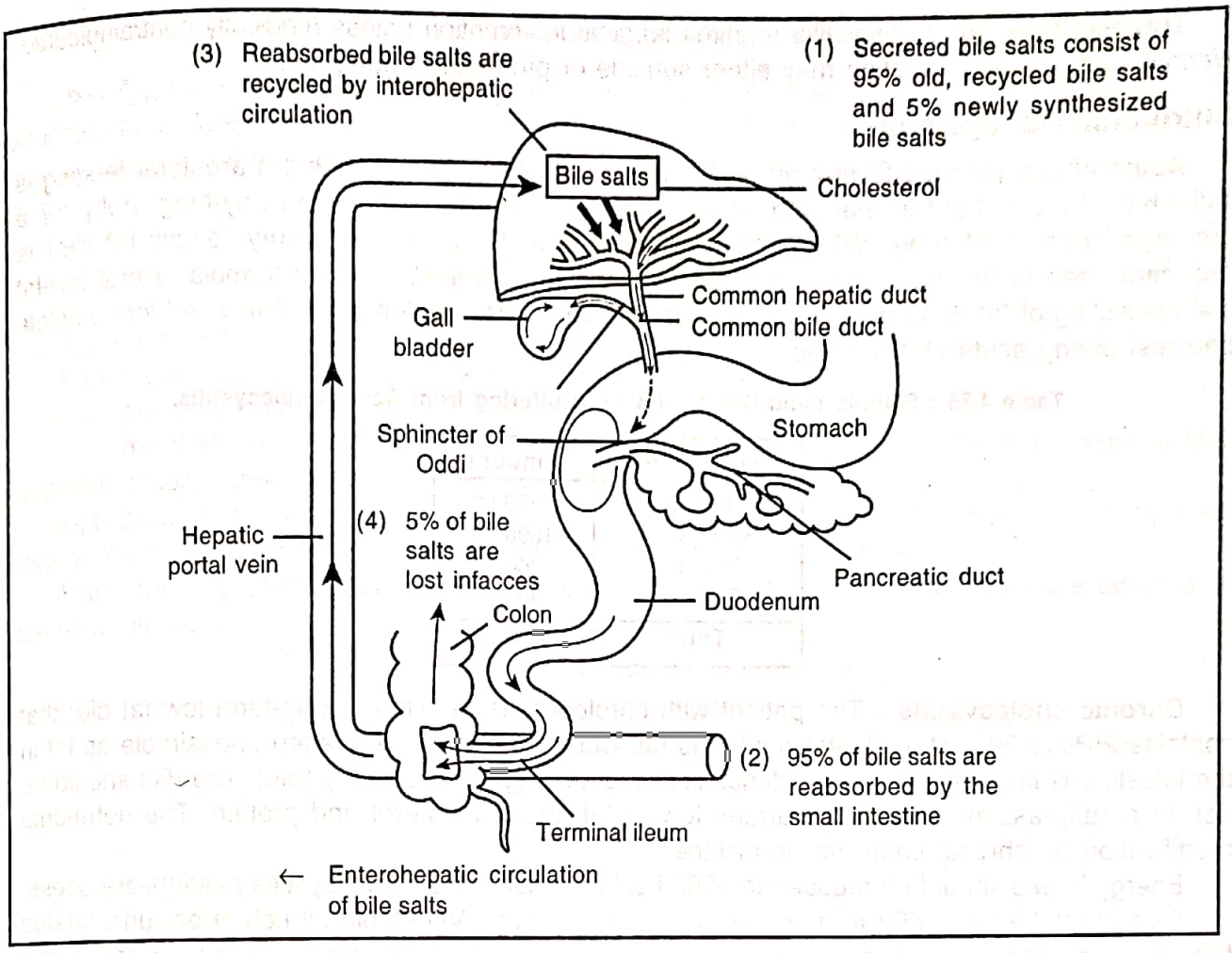

Fig. 4.16 : Enterohepatic Circulation.

DISEASES OF THE GALLBLADDER CHOLECYSTITIS

Inflammation of the gallbladder is known as cholecystitis and cholecystitis may be chronic or acute. Cholecystitis generally develops secondary to gallstones obstruction the bile ducts (calculous cholecystitis) leading to back flow of bile, infection, and ischemia of the gallbladder. When biliary tract is obstructed, then it prevents bile from reaching the intestine, it backs up and returns to the circulation. Bilirubin, the bile tissues, therefore, when it overflows into the general circulation, it causes the yellow skin pigmentation and eye discolouration typical of jaundice.

Acute cholecystitis without stones (acalculous cholicystitis) occurs in critically ill patients or due to bile and galbladder obstruction. Due to decreased spontaneous contractile activity and contractile responsiveness of hormone cholescystokin, the gallbladder emptying is impaired in chronic a calculous choleocystokinin. As a result, the walls of the gallbladder become inflammed and distended and is superimposed with infection. In such conditions, patient experiences upper quadrant abdominal pain accompanical by nausea, vomiting, flatulence and fever.

Chronic cholecystitis is long-term inflammation of gallbladder and it occurs due to repeated mild attacks of acute chalecystitis. This can cause thickening of walls of the gallbladder. The gallbladder begins to shrink and eventually loses the ability to perform its function i.e., concentrating and storing bile. The symptoms increases on eating a high fat diet chronic cholecystitis is common in women than in men, and the incidence increases after the age of 40. The risk factors include presence of gallstones and a history of acute cholecystiris. The symptoms of flatulent dyspepsia consists of feeling of epigastric fullness, immediatety after a fatty meal and biliary colic are common in chronic cholecystitis.

Treatment : Acute cholecystitis requires surgical intervention unless medically contraindicated. Without surgery, the condition may either subside or progress to gangrene.

Nutritional Management

Acute cholecystitis : During an acute attack, oral feeding is withheld. Parenteral feeding is indicated of the patient is malnourished and of the patient is not taking anything orally for a prolonged period of time. When the patient is able to take food orally, a low-fat diet is recommended to decrease gallbladder stimulation. An hydrolysed low fat formula or oral low-fat diet consisting of 30 to 45 g of fat per day can be given. The patient is also advised for complete bed rest during acute cholecystitis.

Table 4.36 : Sample Fluid Diet for Patient Suffering from Acute Cholecystitis.

Composition	Amount
Orange juice	1500 ml
Skimmed milk	1000 ml
Glucose	200 g
Sucrose	200 g
Total	**2500 ml**

Chronic cholecystitis : The patient with chrolecystitis requires a long-term low fat diet that contains 25% to 30% of total kilocalories as fat. But strict restriction is aften undesirable as fat in the intestine is important for some stimulation and drainage of the biliary tract. The diet should be rich in readily assimiable carbohydrates low in fat and cholesterol and protein. The nutritional modification on chronic cholecystitis include :

Energy intake should be reduced to 1500 kcal as most of the cholescystitis patients are obese.

Protein intake 50 to 60g to meet the daily requirements. Vegetable oil rich in polyunsaturated fatty acids should be consumed. Animal fats rich in cholesterol should be avoided vitamins and minerals should be as per ICMR (2020) recommendatons.

Table 4.37 : Sample Menu Plan for a Patient Suffering From Chronic Cholecystitis.

Meal Time	Vegetarian	Non-Vegetarion
Early Morning	Fruil juice - 1 glass	Fruit juice - 1glass
Breakfast	Semolina porridge - 1serving Bread toast with jam - 2 slices Fruits - 1 seving Tea - 1cup	Semolina porridge - 1serving Bread toast with jam - 2 slices Fruits - 1 seving Tea - 1cup
Lunch	Cooked rice - 1 serving Dhal soup - 1 seving Cooked vegetables - 1 serving Curd - 2 cups Whole fruits - serving	Cooked rice - 1 serving Chicken soup - 1 seving Cooked vegetables - 1 serving Curd - 1 cup Whole fruits - serving
Afternon tea	Tea - 1 cup Biscuits - 2 Fruit - 1	Tea - 1 cup Biscuits - 2 Fruit - 1
Dinner	Same as lunch	Same as lunch

CHOLELITHIASIS

Cholelithiasis or gallstones are aggregation of cholesterol pigments, calcium, bilirubinate, calcium, phoshate and proteins in various composition, which leads to formation of radio - opaque or radio lucent solid structure in gallbladder or biliary duct. When gallstone is present in the gallbladder and an inflammation is present, then it causes pain, particularly after eating or after a fatty meal.

Gallstones that pass from the Gallbladder into the common bile duct may remain there may pass into the duodenum with or without symptoms. Choledocholithiasis ooccurs when stones slip into the bile duct, producing obstruction, pain, and cramps. If there is obstruction in the flow o bile into duodeum, cholecystitis may result.

Pathophysiology : The gallstone formation involves several stages :

A genetic and metabolic stage in susceptible individuals

A chemical stage, when cholestherol concentration exceeds the solubilizing capacity of bile (supersaturation), cholesterol can no langer remain dispersed.

A physical state the super saturated bile is neucleated and growth of cholestrol monohydrate begins. Aggregation of microscopic crystals results information of stones.

A symptomatic stage occuring when stone inetiates cholecyslitis and blocks the cystic and common bile duct.

Table 4.38 : Risk factors for Gallstone

For Cholesterol Gallstone	For Pigmented Gallstone
Race (Pima Indian, Mexican american and Scandinavians)	Old age
Old age	Sickle cell anaemia
Family history	Thalassaemia
Obesity	Biliary tract infection
Diabetes mellitus	Cirrhosis
Use of lipid lowering drugs or oral contraceptive drugs	Alcoholism
Inflammatony bowel disease	Long tern parenteral nutrition
Bacterial inection	

Treatment : Treatment of gallstone discase involves cholecystectomy specifically if the stones are numerous, large or calcified. The procedure is either done by traditional open laparotomy or as a less invasive laparoscpic procedure.

The chemical dissolution therapy is done with administration of bile acid, chenodeoxycolic acid ursodeoxycholic acid (Litholytic Therapy) or dissolution by extra corporeal shock wave (ESWL) can also be used.

Nutritional Therapy : A fat restricted low caloric, high fibre balanced diet is prescribed in gallstone patient. The nutritonal modification in cholelithasis include :

Energy : excess calorie intake may be a risk factor for gallstone formation. Gallstones are very common among obese people. Therefore, minimum amount of calories is advised to maintain the normal body weight.

Protein : Excess protein intake can increase biliary cholesterol concentration. So, in normal 50 - 60 gram of protein is recommended to meet the daily requirement.

Lipid : Vegetable oils rich in essential faty acid should be consumed instead of animal fat or other cholesterol rich food. For patient to whom fat does not cause any discomfort, vegetable oil can be given. Fried foods should be avoided. At first, 20 to 30 gram of fat is given, gradually, as the patient become tolerable, 50-60 grom of fat is given daily.

As cholesterol can be formed carbohdrate, protein and fat metabolism in the liver. Therefore, there is no need to restrict dietary cholesterol. But reducing the amounts of egg and organ meats in diet can be considered as preventive measure.

Carbohydrate : Intake of excess simple sugars in beverages and sweets is associated with formation of gallstone.

Dietary Fiber : Fiber plays an important role in preventing gallstone. Fiber interacts with bile salt, insoluble fiber after combination of bile acid alter the composition bile acid poll, increases the level of chenodeoxycholic acid, enhances the biliary cholesterol solubility. So, a high fiber diet should be recommended.

Vitamins and Minerals : As a low fat diet is presribed in cholelithiasis, supplemertation of sholuble vitamins A, D,E,K is necessary. recquirement should be as per ICMR (2020) recommendation.

Fluids : An excess fluid intake advised or gallstone patients.

Table 4.39 : Foods to be Included and Avoided in Cholelithiasis.

Food to be Included	For to be avoided
Bread, chapaties	Fruits dried
Wheat, Oat meals	Nut
Rice cooked	Sweet and sweet meals
Pulses, beans	Condiments and spices
Veg salad cooked patatoes soup	Papad, Chuntney, prickles
Meat, fish except organ meat	Organ meat
Milk and milk products	Egg
Sugar, Jam	
Pastry, Biscuit	
Fruits	
Beverages	
Fluid liberal	

Table 4.40 : Sample Menu Plan for a Patient Suffering Cholelithisis.

Meal Time	Vegetarian	Non-Vegetarion
Meal Time Breakfast	Orange - 1 glass Porridge with skim milk - 1cup Toasted bread - 1 slice Tea - 1 cup	Orange Juice- 1 glass Porridge - 1bowl Toasted bread with jam- 1 slice Tea - 1 Cup
Mid - Morning	Tea - 1 cup Biscuits - 2 Apple baked - 1	Tea - 1 cup Biscuits - 2 Apple baked - 1
Lunch	Cooked Rice - 2 cup Veg. Dal - 1 cup Veg. currry - 1 bowl Soybean or paneer curry - 1 bowl	Rice cooked - 2 cup Veg. Dal - 1 cup Veg. curry - 1 bowl Soybean or paneer curry - 1 bowl Fish curry - 50gm.
Afternoon Tea	Tea - 1 cup Biscuits - 3	Tea - 1 cup Biscuits - 3
Dinner	Min, veg soup - 1 bowl Mashed potato - 1 Chapati - 2 pcs Green pea curry - 1 bowl Curd - 1 cup	Chicken soup with vegetable - 1 bowl Chapati - 2 pcs Green pea curry - 1 bowl Cucumber Raita - 1 bowl

DISEASE OF EXOCRINE PANCREAS

PANCREATITIS

Pancreatitis refers to the inflammation of the pancreas and is characterized by oedema autodigestion, cellular exudates, fat necrosis and haemorrhage of pancreatic tissue. This disease can range from mild to self limiting to severe condition which results in autodigestion necrosis and haemorrhage of pancreative tissue.

Pancreatitis can be eighter acute pancreatitis or chronic pancreatitis.

Acute pancreatitis are due to formation of gallstone (including microlithiosis), acute and chronic alcoholism, hyper triglyceridaema, trauma, post operative condition (abdominal and non abdominal operation), dysfunction of sphincter of Odi and several drug administration (Azathioprine), 6-mercaptopurine, sulfanamides, oestrogens, tetracycline, valproic acid, and anti HIV medication).

General pathology : Bile is synthesized by the liver stored in the gall bladar and secreted in the intestine. If has distinct role in fat metabolism and decresed production and secretion of bile can affect fat digestion leading to fat malabsorption. A building of back up bile pressure due to renal or post renal causes can lead to specification of jaundice can be a cause of secondary biliary cirrohosis and cholelithiasis. Obstruction of distal common bile duct can lead to pancreatitis if the pancreatic duct is blocked. Thus, liver gallbladder and pancreative diseases can have an overlapping aetiology. The exact mechanism that can lead to pancreatic injury is not fully understood. However a common characteristies seems to be premature activation of trypsin with thin the pancreas resulting in auto digstion of pancreatic cells. The enzymes released by pancreatic cells evantually reach the blood stream, thereby causing elevation in serum amylase and lipase levels.

Clinical Features : While some cases of acute pancreatitis remain asymptomatic, common symptoms may include upper abdominal pain radiating to the back, generally worsening with in gestion of food. The other symptoms include nausea, vomiting, abdominal distension and steatorrhoea. In severe conditions, hypotension and dehydration may occur.

The clinical features chronic pancreatitis include chronic abdominal pain and normal to mild elevation of panceatic enzyme levels. In addition, steatorrrhea with loss of pancreatic endocrine and exocrine function are also found. Jaundice may be present.

Treatment :

Acute pancreatitis : Pain assoiated with pancreatitis should be treated accordingly and vomiting can be controlled by removing the gastric content contionnously.

Chronic pancreatitis : Blood treatment transfusion, fluid and electrolytes management, antibiotic therapy and pain releif medication are advised in chronic pancreatitis.

Nutritional Managemant : Patients of pancreatitis are commonly malnurished as a result of : poor intake due to anorexia and severe pain; malabsorption of nutrients consumed; due to increased requirement in catabolic states and for the frequent episodes of nil by mouth during treatment.

So, provision of adequate nutritionl support is essential for these patients. Planning a planning for a diet a pancretitis patent is a skilled job should be recommended by a registered dietitian.

Acute pancreatitis : Pain assoiated with acute pancreatitis is partially related to secretory mechanism of pancreatic enzyme and bile. So, nutritional therypy should be adjusted to provide minimum stimulation of these systems.

During an acute attack, all foods by mouth is withheld and hydration is maintained through intravenous feeding. In less severe attacks, a clear fluid diet with restricted fat is given or the days. The patient should be monitored for any symptom such as pain, nausea or vomiting. There after the patient is progressed as tolerated to easily digested food, to with low fat content. Instead of heavy large meals, the food should be divided into six small meals for better toleration, Patients with mild to moderate stress can tolerate dextrose based solution, where as patients with more severe stress reguire a mixed fuel system of dextrose based solution. where as, patients with

move severe stress reguire a mixed fuel system of dextrose and lipid to avoid complications of glucose in tolerance.

Nasogastrc feeding is feasible in move then 80% of patients.

In contrast to acute pancreatitis chronic pantent is pancreatitis is characterised by recurrent attacks of epigastric pain of long duration that radiates into the back. The goal of nutritional therapy of patients of chronic pancreatitis to prevent further damage to the pancreas, decrease the number of attack of acute inflammation, aleviate pain, decreased steatorrhoea and correct malnutrition. The frequency of attack may be reduced by advising frequent, small meals of a moderate to low fat diet.

Pancreatic enzymes are prescribed to be taken at each meal may vary depending on the fat content of the food consumed.

Substitution of dietary fat with MCT (medium chain triglycerides) may relief steatorrhoea and lead to wight gain as MCTs do not require lipase for digestion. Supplementation of vitamins and minerals is the commended. Water soluble forms of fat soluble vitamins and parenteral adninistration of vitamin B_{12} may be necessary. Dietary recommendations should be adjusted for other pancreatits, such as diabetes, obesity and alcoholism. Approximately, one third of patient with chronic pancreatitis will develop diabetes mellitus; a compromise should be reached between dietary advise to optimize blood sugar and to enhance intake to maintain body weight or reverse weight loss. For those patients with a history of alcoholism thiamine (100 mg orally once a day) and folate (1mg by month once a day) should be advised obstaining from alcohol is advisable a continuing alcoholism is associated with increased morbidity & mortality.

Table 4.41 : Sample Menu Plan for a Patient Suffering from Chronic Pancreatitis.

Meal Time	Vegetarian	Non-Vegetarion
Early Morning	Fruil juice - 1 glass	Fruit juice - 1glass
Breakfast	Mike semolina - serving Fruits - 1 seving Tea - 1cup	Mike semolina - serving Fruits - 1 seving Tea - 1cup
Mid morning	Fruit juice - 1 glass	Fruit juice - 1 glass
Lunch	Cooked rice or Roasted bread - 1 serving Vegetab soup - 1 seving Cooked tender vegetables - 1 serving Curd - 1 cups Fruit - 1 serving Skim milk pudding - 1 serving	Cooked rice or Roasted bread - 1 serving Vegetab soup - 1 seving Fish curry - 1 serving Curd - 1 cups Fruit - 1serving Skim milk pudding - 1 serving
Afternon tea	Tea - 1 cup Biscuits - 2 Fruit - 1	Tea - 1 cup Biscuits - 2 Fruit - 1
Dinner	Same as lunch	Same as lunch

Pancreatitis

Pancreatitis is a disease in which the pancreas becomes inflammed. (Pancreatic damage happens when the digestive enzymes are activated before they are released into the small intestine and being attacting the ~~pancreas~~ pancreas.)

Types :—

Acute pancreatitis :— It is sudden inflammation that last for a short time. It may range from mild discomfort to a severe life threatening illness. Most of the people with acute pancreatitis recover completely after getting right treatment. In

severe cases acute p can result in bleeding into the gland, serious tissue damage, infection, and cyst formation.

Chronic Pancreatitis :—

It is long lasting ~~discomfort~~ inflammation of the pancreas. It most often happens after an episode of acute pancreatitis. Heavy alcohol drinking is ~~the~~ another big cause. Damage to the pancreas from heavy alcohol use may not cause symptoms for many years but then the person may suddenly develop severe pancreatic symptoms.

Pathophysiology :—

1) Activation of P. Enzyme inside the pancreas cause damage.

2)

In normal ~~condition~~ digestion the inactivated p. enzyme move through p. ducts travel to the s. i, while the enzymes becomes activated & help wd digestion

In pancreatitis the enzyme becomes activated while still in the pancreas. This causes the enzymes to irritate the cells of pancreas, causing inflammation & the signs and symptoms associated wd pancreatitis.

Wd repeated attack of acute pancreatitis, damage to the pancreas can occur & lead to chronic pancreatitis. Scar tissues may form in pancreas, causing loss of function. A poorly functioning pancreas can cause digestion problems & diabetes mellitus.

A no of causes have been identified for acute pancreatitis & chronic pancreatitis. Includes
→ Alcoholism
→ Gall stone
→ Abdominal surgery.

Acute → is caused by gall stones or heavy alcohol use. Other cause include infection, medication, trauma, metabolic disorder & surgery. Up to 15% of people wd acute pancreatitis is unknown.

Chronic :— 70% of people, chronic is caused by long-term alcohol use. Other cause gall stones, hereditary disorder of - pancreas, cystic fibrosis, high TG, certain medicine, 20—30%. Case is unknown.

Risk factor

It happens to any one but most common in people who certain risk factors —

Risk factor for acute pancreatitis

→ Gall stones
→ Heavy alcohol drinking:

Gall stones can block the pancreatic duct, ~~which is the cause of acute p~~ stopping p. enzyme from travelling to the s. intestine where enzyme start to irritate cells of pancreas, & causing pancreatitis.

Risk factor for

Acute chronic :—

→ heavy alcohol intake
→ certain hereditary conditions such as cystic fibrosis.
→ Gall stone
→ high TG.

people wd chronic pancreatitis are usually men between age 30 & and 40, chronic pancreatitis also may occur in women.

symptoms

Acute :→ upper abdominal pain that radiate to the back & may be aggravated by eating specially foods high in fat.
→ swollen & tender

Sunday 08

abdomen.
→ Nausea & vomiting
→ fever
→ increased heart rate
→ jaundice → severe back

Symptoms of chronic pancreatitis

Same as acute,
→ patient frequently feel constant pain in the upper abdomen that radiate to back

other symptoms
→ wt loss caused by malabsorption of fat. due to unavailable p. enzyme which helps in digestion of fat.

→ diabetes mellitus may develop & cell of

Pancreas are damaged

→ steatorrhoea
→ jaundice
→ Enlarged gall bladder & liver

Complications

→ pseudo cyst
→ Infection
→ Breathing problems - can cause chemical change in body affecting lungs ↓ O_2, unable to flow.
→
→ Diabetes
→ kidney failure
→ malnutrition
→ pancreatic cancer.

Diagnosis :—

-) measurement of amylase & lipase - the two digestive enzyme. high level ~~may~~ strongly suggest A.P.

-) Pancreatic function test to find out if the pancreas is making the right amt of digestive enzyme.

-) Glucose tolerance test to measure the damage to the cell in the pancreas that make ~~[illegible]~~ insulin

-) Ultrasound, CT scan, MRI to make image.

-) Biopsy.

shallow started immediately.

hospitalization

Treatment

→ Acute → Typically treated wd IV fluid & pain killer. in hospital.

→ Severe → ICU, closely watched.

→ Surgery to remove org if infected.

→ Removal of gall stone gallbladder surgery of bile duct.

Chronic (difficult - lot of no)

→ Relieve Pain

→ Impaired nutrition problem

→ Insulin

→ Low fat diet.

C.P

Nutritional support :-

high Δ (35 kCal/kg/bw)

hi ll Protein (1 - 1.5 g/kg)

↑ CHO

~~moderate~~ low fat (0.7 - 1.0 g/kg)

→ If diabeter is present then ↓ simple CHO.

Normally

② veg fat is better tolerated than animal fat.

→ MCT must be used ~~to~~

it to increase fat absorption bec- they need minimal digestion and donot require lipase, colipase, & bile salts.

→ MCT should be increased slowly ~~acc~~ according to the tolerance of the patient.

→ ~~Diet sho~~ Oral nutrition therapy should be low in ~~B~~ ~~fat~~ for it delay absorption of nutients.

→ A, D, E, K, B12, antioxidants

should be replaced as clinically indicated.

→ Ca & vit D supplementation with physiologic dosage range are recommended for vit D deficiency. e.g. even in the case of pancreatic calcification.

Lipase supplements reduce steatorrhoea in CP.

→ Jejunal feeding improves Wt, reduces nacrotic syndrome, & improves patient's quality of life.

To keep pancreas healthy. High chol, ↑ Cal and ↑ consumption of alcohol should be avoided.

Sunday 15

acute

~~parenteral~~ parenteral administration of 2L to 2.5 L of 10% glucose (if blood sugar is normal) to provide minimal amounts of calories should be done.