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GALLSTONE DISEASE AND THE ROLE OF DIET TABLE

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Abstract. Gallstone disease (GSD) is one of the most common disorders of the hepatobiliary system and is characterized by the formation of stones in the gallbladder and bile ducts. The disease develops as a result of disturbances in cholesterol, bilirubin, and bile metabolism. This article reviews the etiology, pathogenesis, risk factors, clinical features, and dietary management of gallstone disease, with special emphasis on the role of Diet Table No. 5 in patient care.

Keywords: Gallstone disease, cholelithiasis, gallbladder, bile ducts, cholesterol metabolism, diet therapy, Table No. 5 diet.

Introduction. Gallstone disease is the formation of bilirubin, cholesterol, and other types of stones in the gallbladder (cholecystolithiasis), the common bile duct (choledocholithiasis), or the intrahepatic bile ducts (intrahepatic cholelithiasis). This disease usually occurs in older adults, particularly in women with disorders of fat metabolism and excess body weight. Gallstone disease (GSD) is a group of disorders that develop as a result of metabolic changes in the hepatobiliary system and are characterized by the formation of gallstones due to disturbances in cholesterol and bilirubin metabolism. If these stones form in the gallbladder, the condition is called cholecystolithiasis (gallbladder stones); if they form in the common bile duct, it is called choledocholithiasis; and if they form in the intrahepatic bile ducts, it is referred to as intrahepatic cholelithiasis. The disease is manifested by pain syndromes caused by biliary obstruction, smooth muscle spasm, and increased pressure within the bile ducts.

Various metabolic disorders, particularly the increasing number of obese individuals, contribute to the growing prevalence of gallstone disease among the population.

In most cases, gallstone disease develops in patients who have several predisposing risk factors. Obesity, physical inactivity, age over 40 years, fair hair color, hereditary predisposition, biliary tract dyskinesia, use of estrogen-containing hormonal medications, female sex, elevated levels of high-density lipoproteins, and increased triglyceride levels, as well as certain associated diseases (diabetes mellitus, familial hyperlipoproteinemia), are included among these risk factors.



Picture 1. Gallstones in the gallbladder

Etiology: Primary bile acids are synthesized from cholesterol in the liver. Vitamin C promotes the conversion of cholesterol into bile acids through hydroxylation. These primary bile acids are converted into secondary bile acids in the intestine. Tertiary bile acids are further modified from secondary bile acids by intestinal flora or hepatocytes. Bile acids are soluble, with a hydrophilic hydroxyl group, a glycine or taurine side chain, and a hydrophobic steroid ring. Gallstones are formed from bile products that precipitate out of solution, including cholesterol, breakdown products of red blood cells, and a mixture of calcium bilirubinate, phosphate, carbonate, palmitate, and cholesterol. These products are suspended in a mucin glycoprotein matrix that acts as a nucleating factor for stone formation. Additional substances such as prostaglandins and arachidonyl lecithin

promote stone crystallization.

Cholesterol is the main component of the most common type of gallstone. Black stones, composed of calcium bilirubinate, result from the breakdown of hemoglobin. In contrast, brown stones form in the setting of bacterial or parasitic infection and are composed of a combination of calcium substrates, including calcium bilirubinate, calcium phosphate or palmitate, cholesterol, and bile. Cholesterol stones are prevalent in individuals with diabetes and other metabolic dysfunctions. In contrast, black stones occur in those with an inflammatory disease, such as Crohn disease, or in those who undergo hemolysis, and brown-pigmented stones are observed in those with parasitic or bacterial infections and biliary strictures. Primary gallbladder stones remain within the gallbladder or migrate into the cystic and common bile ducts, potentially causing pain and obstruction; gallstones may also form primarily within the ductal system.

Pathophysiology. Gallstones occur when substances in bile exceed their solubility. As bile becomes supersaturated, small crystals precipitate and become stuck in gallbladder mucus, resulting in gallbladder sludge. Over time, these crystals coalesce and form large stones. The stones are often mobile and can migrate into the bile ducts. Cholangitis and pancreatitis may result if the biliary system becomes obstructed. Stones composed of calcium bilirubinate may also form primarily in the ducts, whereas stones formed in the gallbladder are predominantly cholesterol stones. Gallstones are associated with cirrhosis and form in the setting of biliary stasis from, for example, spinal cord injury and gastrectomy, and medications such as somatostatin and estrogen. Study results show that gallbladder innervation is diminished in those with gallstones. Cholesterol stones result from increased bile cholesterol due to hepatic secretion and excess triglycerides. These factors promote the secretion of gallbladder mucin, contribute to a larger fasting gallbladder volume, reduce postprandial emptying, and impair intestinal motility. Excess biliary cholesterol can also cause gallbladder wall proliferation and inflammation. Medications, phospholipid homeostasis, gut microbiome, diet, metabolic syndrome, hormonal and bacterial milieu, genetics, and even high altitude contribute to cholesterol gallstone formation. Fructose promotes gluconeogenesis and lipogenesis. Once glycogen accumulates in the liver, glycogen intermediates are used for triglyceride synthesis, and hypertriglyceridemia can cause decreased gallbladder emptying and impaired contractility due to decreased sensitivity to cholecystokinin.

Cholesterol transporters can influence the amount of cholesterol in blood or bile. Lecithin increases cholesterol concentration in bile carried in lecithin vesicles that fuse and form cholesterol hydrate crystals, which become the nucleus of a cholesterol stone. Bile composition influences the rate of cholesterol crystallization, and a hypoactive gallbladder allows more time for crystals to form. Phospholipid lamella also transports excess cholesterol and plays a role in cholesterol crystalization in conjunction with prostaglandins and arachidonyl lecithin. Granulocytes and

neutrophils are attracted to the cholesterol crystals, and neutrophils shed deoxyribonucleic acid onto the crystals, which attract similar crystals, thus forming larger stones.

Treatment. Treatment for gallstones ranges from reassurance to dietary and lifestyle recommendations to referral for operative management. Nonoperative management of biliary colic involves strict maintenance of a low-fat diet and supportive treatment with antiemetics and pain control, and counseling regarding risk for recurrence. Those with repeat episodes of colic are referred for an elective laparoscopic cholecystectomy. However, suppose the patient has severe abdominal pain or cannot tolerate oral intake. In that case, hospital admission and more urgent surgical management are warranted for symptomatic relief and to prevent further

clinical sequela.

Impacted stones will cause progressive and unrelenting pain and are an indication for admission and timely surgery. Stones greater than 1 cm in size have a greater propensity to obstruct the cystic duct and may predispose to acute cholecystitis, so they should be treated with surgical management more urgently. Laparoscopic cholecystectomy is the standard of care for symptomatic gallstones. Open cholecystectomy is performed when the laparoscopic approach is not feasible or is found to be too difficult intraoperatively. In cases of acute cholecystitis in unstable patients or patients with poor surgical candidates, a cholecystostomy tube can be placed by interventional radiology as either a temporizing or palliative measure.

Conclusion. Gallstone disease (GSD) is a common disorder of the hepatobiliary system characterized by the formation of stones in the gallbladder or bile ducts due to disturbances in cholesterol, bilirubin, and bile metabolism. The disease is more frequently observed in older adults, especially in women with obesity and lipid metabolism disorders. Early diagnosis and proper management are important to prevent complications and improve patients' quality of life.

Diet therapy plays a significant role in the treatment and prevention of gallstone disease. Diet Table No. 5 is recommended because it helps restore liver and biliary tract function, improves bile secretion, and reduces the risk of stone formation.

General Characteristics. A physiologically normal diet containing adequate amounts of proteins and carbohydrates, with a moderate restriction of fats (especially refractory fats). Foods rich in nitrogenous extractives, purines, cholesterol, oxalic acid, and essential oils are excluded.

The amount of lipotropic substances, fiber, pectins, and fluids is increased. Foods are prepared by boiling or baking. Tough meat, high-fiber vegetables, fresh flour products, and very cold dishes are excluded.

Chemical Composition and Energy Value: Proteins: 90–100 g (60% of animal origin) Fats: 80–100 g (30% vegetable fats) Carbohydrates: 400–450 g (70–80 g sugar) Energy value: 11.7–12.2 MJ (2800–2900 kcal) Sodium chloride: 10 g Fluids: 1.5–2 liters per day Dietary Regimen: Meals should be taken 5 times a day.

Foods and dishes not allowed: fresh hot bread, puff pastry, fried meat, rich meat broths, fish broths, mushroom broths, fatty meat, duck meat, goose meat, liver, kidneys, brains, smoked sausages, canned foods, fatty fish, salted fish, hard-boiled eggs, fried eggs, beans, legumes, spinach, sorrel, radish, green onions, garlic, mushrooms, pickled vegetables, caviar, chocolate, cream products, ice cream, mustard, pepper, black coffee, cocoa, cold beverages

References:

1. Sh.S.Narziyev. Ichki kasalliklar propedevtikasi 2018
2. A.G.Gadayev. Ichki kasalliklar propedevtikasi 2016

3. E S, Srikanth MS, Shreyas A, Desai S, Mehdi S, Gangadharappa HV, Suman, Krishna KL. Recent advances, novel targets and treatments for cholelithiasis; a narrative review. Eur J Pharmacol. 2021 Oct 05;908:174376. [PubMed] [Reference list]
4. Hapca S, Ramsay G, Murchie P, Ahmed I. Biliary colic. BMJ. 2021 Sep 13;374:n2085. [PubMed] [Reference list]
5. Shabanzadeh DM. New determinants for gallstone disease? . Dan Med J. 2018 Feb;65(2) [PubMed] [Reference list]
6. Cianci P, Restini E. Management of cholelithiasis with choledocholithiasis: Endoscopic and surgical approaches. World J Gastroenterol. 2021 Jul 28;27(28):4536-4554. [PMC free article] [PubMed] [Reference list]
7. Di Ciaula A, Wang DQ, Portincasa P. An update on the pathogenesis of cholesterol gallstone disease. Curr Opin Gastroenterol. 2018 Mar;34(2):71-80. [PMC free article] [PubMed] [Reference list]
8. E S, Srikanth MS, Shreyas A, Desai S, Mehdi S, Gangadharappa HV, Suman, Krishna KL. Recent advances, novel targets and treatments for cholelithiasis; a narrative review. Eur J Pharmacol. 2021 Oct 05;908:174376. [PubMed] [Reference list]
9. Sun H, Warren J, Yip J, Ji Y, Hao S, Han W, Ding Y. Factors Influencing Gallstone Formation: A Review of the Literature. Biomolecules. 2022 Apr 06;12(4) [PMC free article] [PubMed] [Reference list]
10. Mark W.Jones, Connor B.Weir, Mia Marietta. Gallstones(Cholelithiasis). 2025 June;2 PMID: 29083691