MINI REVIEW

Pathophysiology, Complications, Diagnosis and Treatment of Gallstone Disease

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ABSTRACT

The development of mouse models of gallstones, their significance to genetic studies of gallstone disease, and the most recent breakthroughs in gallstone pathophysiology from animal experiments are summarized. Physical-chemical, genetic, and molecular biological studies of gallstone disease in mice with knockout or transgene of specific target genes have revealed many novel insights into the intricate pathogenic processes of this very common hepatobiliary disease, demonstrating that interactions of five primary defects play an important role in the pathogenesis of cholesterol gallstones. Based on mice research, it has been postulated that several Lith genes generate hepatic hypersecretion of biliary cholesterol, with insulin resistance as part of the metabolic syndrome interacting with cholelithogenic environmental variables.

INTRODUCTION

Obesity and Cholesterol Gallstone Disease (GSD) are commonly comorbid disorders; consequently, given the current global obesity pandemic, a thorough understanding of the pathophysiological links between GSD and insulin resistance (IR) is critical. Obesity was formerly thought to be a risk factor for GSD, and the gallbladder (GB) was thought to be only a bile reservoir with no metabolic functions. Consistent data suggests that both GSD and cholecystectomy are associated with fatty liver and IR, raising the notion that the GB is a metabolic regulatory organ. We discuss the pathophysiological mechanisms that link GSD, IR, and obesity, with a focus on the GB's functions as a regulator of bile acid kinetics and a hormone secreting organ with systemic metabolic actions. We also investigate the links between enhanced hepatic lipogenicity in IR conditions and GSD development [1].

CAUSES OF GALLSTONES

Acute pancreatitis is one of the most common gastrointestinal reasons for hospitalization. Although chronic pancreatitis is less common, it has a significant impact on patients' quality of life. Pancreatic cancer has a high mortality rate and is one of the top five cancer-related causes of death. The prevalence of pancreatic disorders is expected to rise in the coming years. Pancreatitis risk and aetiology vary with age and gender, and all pancreatic disorders affect the black population more than any other race. Gallstones are the most common cause of acute pancreatitis, and cholecystectomy prevents future attacks. The single most important health risk for chronic pancreatitis remains alcohol. Smoking is a risk factor for both acute and chronic pancreatitis, and its effects may interact with those of alcohol. Smoking and non-O blood groups are significant risk factors for pancreatic cancer. Smoking cessation and alcohol abstinence can slow the progression of pancreatitis and reduce recurrence; smoking cessation is the most effective strategy for lowering the risk of pancreatic cancer [2].

TREATMENT

The most significant methods for gallstone disease prevention are regular physical exercise and a healthy diet. Transcutaneous ultrasonography is the gold standard for detecting gallstones. Endoscopic retrograde cholangiography should only be conducted as part of a planned therapeutic intervention; doing endosonography prior reduces the number of endoscopic retrograde cholangiographies required. Cholecystectomy is recommended for people who have symptomatic gallstones or sludge [3].

If possible, this should be done laparoscopically using a four-trocar method. Antibiotic prophylaxis during

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surgery is not required. If necessary, a cholecystectomy can be performed throughout any trimester of pregnancy. Acute cholecystitis requires immediate laparoscopic cholecystectomy within 24 hours of hospitalisation. After successful endoscopic biliary route clearance, patients with cholelithiasis should have a laparoscopic cholecystectomy [3].

RISK FACTORS

Despite substantial research into cholelithiasis risk factors, several studies reveal that definitive results remain difficult. This study identifies risk factors for cholelithiasis, describes the pathophysiology of gallstone disease, and discusses nonsurgical preventive approaches. Understanding the risk factors for cholelithiasis may aid nurses in not only providing resources and information to patients with gallstones, but also in creating novel preventive methods for the disease [4].

MORTALITY RATE

The goal of this cohort study was to see if patients with gallstone disease diagnosed through general population screening had higher overall mortality than gallstone-free participants and to look into causes of death. Gallstone disease was substantially related with overall mortality and death from cardiovascular illnesses. Gallstone disease was substantially related with death from unknown causes, while cancer and gastrointestinal disease were not. There were no differences in mortality for ultrasound-proven gallstones or cholecystectomy. Gallstone disease is linked to an increase in overall mortality as well as death from cardiovascular disease. Gallstones could be a risk factor for cardiometabolic disease. Other unknown elements appear to be at work as well [5].

CONCLUSION

The mouse gallstone model is critical for studying the physical-chemical and genetic mechanisms of cholesterol crystallization and gallstone development, which improves our understanding of the aetiology of this illness in humans. We propose a model in which GSD and hepatic IR interact to produce a state of dysregulated lipid and energy metabolism that exacerbates the metabolic dysregulation caused by obesity.

References

1. Cortés VA, Barrera F, Nervi F. Pathophysiological connections between gallstone disease, insulin resistance, and obesity. Obes Rev. 2020;21:e12983. [PMID: 31814283].

2. Gutt C, Schläfer S, Lammert F. The treatment of gallstone disease. Dtsch Arztebl Int. 2013;144:1252-1261. [PMID: 31814283].

3. Cai JS, Qiang S, Bao-Bing Y. Advances of recurrent risk factors and management of choledocholithiasis. Scand J Gastroenterol. 2017;52:34-43. [PMID: 27610642].

4. Pak M, Lindseth G. Risk factors for cholelithiasis. Gastroenterol Nurs. 2016;39:297-309. [PMID: 27467059].

5. Shabanzadeh DM, Sørensen LT, Jørgensen T. Gallstone disease and mortality: A cohort study. Int J Public Health. 2017;62:353-360. [PMID: 27815564].

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